"A Histopathological Study of the Nasal Mucosa Pre and Post Anti-Allergic Medication Including Oral and Topical Drugs in Allergic Rhinitis in Bundelkhand"

THESIS
FOR

DOCTOR OF SURGERY
(OTOLARYNGOLOGY)

BUNDELKHAND UNIVERSITY,
JHANSI (U.P.)

2005 **JITENDRA SINGH YADAV**

Dedicated to My Parents

CERTIFICATE

This is to certify that the work entitled **"A Histopathological Study of the Nasal Mucosa Pre and Post Anti-Allergic Medication Including Oral and Topical Drugs in Allergic Rhinitis in Bundelkhand"** which is being submitted as a thesis for M.S. (E.N.T.) Examination 2005 of Bundelkhand University, Jhansi, has been carried out by **Dr. Jitendra Singh Yadav** in the Department of E.N.T., M.L.B. Medical College, Jhansi.

The method described was undertaken by the candidate himself and the observations recorded have been periodically checked. He has put in the necessary stay in the Department as per University regulations, and has fulfilled the conditions required for the submission of thesis according to University regulations.

Dated:

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M.S., E.N.T.,
Professor & Head,
Department of E.N.T.
M.L.B. Medical College,
Jhansi.

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Dated:

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Date :

Jitendra Singh Yadav

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Introduction

INTRODUCTION

"A Histopathological study of the nasal mucosa pre and post antiallergic medication including oral and Topical Drugs in Allergic Rhinitis in Bundelkhand".

The Allergic Rhinitis is a common cause of morbidity, social embarrassment and impaired performance either at school or work place. It is defined as IgE mediated hypersensitivity disease of mucous membrane of nasal airway characterized by sneezing, itching, watery nasal discharge and nasal obstruction. It may be associated with Allergic Conjunctivitis and Bronchial Asthma.

- The disease has been found to have some relation with age and sex.
- The disease is common in certain seasons of the year.

Aetiology

1. Atopy
2. Seasonal Rhinitis
3. Perennial Allergic Rhinitis
4. Occupational Allergens
5. Food and Drug induced Rhinitis
6. Role of pollution

Atopy refers to the tendency to develop an exaggerated IgE antibody response as reflected by a positive skin prick test in response to one or more common aeroallergens. Atopy affects approximately 1/3 of the population but the manifestation of the disease does not occur in all people. Atopy is genetically inherited but mode of inheritance may be multifactor. The recent study shows that the gene which has high affinity to IgE receptor is present on 11q chromosome.

Seasonal Allergic Rhinitis in the U.K. is most commonly due to allergy to grass pollen with seasonal symptoms in June or July corresponding to peak of grass pollen counts, In India, In Shivpuri & his colleagues in Delhi & Kabliwal in Jaipur were the pioneers in the work of allergy. Shivpuri's groups established that the common allergens are Helianthus, Amaranthus, Cassia, Cenchrus, Morus pollen. The pollen counts above 50/ m^3 are considered to be the level at which most hay fever sufferers may develop symptoms. Seasonal Allergic Rhinitis may also occur due to exposure of tree pollens, weed pollens, fungi spores. Paradoxically rainfall may provoke an initial increase in spore numbers and resulting of epidemic of Asthma and Rhinitis.

The commonest cause of **Perennial Allergic Rhinitis** is allergy to house dust mite. Mite species include D. pteronysinnus, D. farinae, Euroglyphus maynei in decreasing order of frequency but in India D. farinae is most common. In Jhansi district, the parthenium hysterophones pollen was main allergen. In pethenium pollen occurs in large clump formation. The optimum conditions for mite growth are approximately 15-20° C and relative humidity 60-70%. The major allergens of house dust mite have been identified as digestive enzymes (Cysteine protease, group 1 allergens eg Der pi) present in the digestive tract and excreted in mite faeces .The sources of other major perennial allergens are domestic pets (cats, dogs, rabbits and guinea pigs).

Occupational Rhinitis is far less well characterized. The common biological causes include flour (in bakers, grain worker) wood dusts, biological washing powder and colophony. Recently identified allergen is latex in surgical gloves which may produce intraoperative life threatening anaphylaxis.

Food may occasionally provoke IgE mediated allergic rhinitis. It may produce nasal symptoms as well as symptoms in other organs including mouth, tongue and digestive tract. The food induced rhinitis may be due to sensitivity to its preservative like sulphide, benzoate or tartrazine. Histamine -containing food such as cheese, poorly kept fish and certain wines may provoke pseudo allergic reactions including flushing, headache and rhinitis. Alcohol may exaggerate nasal congestion in Allergic Rhinitis person. Milk, egg and cheese allergy are more common in children while citrus fruit, nut and shellfish allergy in adult .Drugs (Aspirin and Anti - hypertensive drugs) may produce rhinitis.

Rhinitis medicamentosa refers to rebound hyperemia, nasal congestion, and obstruction with tachyphylaxis that occurs following prolonged and repeated use of topical vasoconstrictors .

The role of pollution is controversial. Non specific, irritants may heighten the sensitivity of nasal mucosa known as idiopathic rhinitis.

Pathogenesis

The characteristic feature of atopy is the preferential production of IgE antibodies by human B Lymphocytes in response to antigenic stimulation by common aeroallergens. The high Affinity IgE receptors (Fc epsilon RI) are present on mast cells, basophils. Langerhans cell and eosinophils have low affinity IgE receptors (Fc epsilon RI) have been demonstrated on T lymphocytes, B lymphocytes, monocytes and macrophages, eosinophils and platelets.

The CD^{4+} lymphocytes are essential for IgE production by B cells. In recent years, two functionally different populations of CD^{4+} helper cells

have been recognized. The helper-1, (Th1) subset synthesizes and secretes IL-2 and IFN-γ where as Th2 cells produce 1L-4 and 1L-5. CD^{4+} Th2 cells and Mast cells produce IL-4 and IL-5 and are responsible for IgE production.

While 1FN- y inhibit the 1L-4 hence decreases production of IgE by B cells. The Allergen - IgE dependent activation of Mast cells and basophils result in production of whole range of pharmacologically active mediators. Translocation of cytosolic granules to the cell surface occurs and where membrane exposes the granular matrix to the extra cellular environment and release mediators like histamine, tryptase, bradykinin. In Parallel with this event membrane phospholipases (C and A2) are stimulated which act on membrane phospholipids and produces arachedonic acid. The metabolism of arachedonic acid occurs in two pathway.

1. Cyclo-oxygenase pathway generates predominantly PGD_2.
2. lipoxygenase pathway generates leukotrienes B_4, C_4 and D_4 known as slow releasing substance of anaphylaxis (SRS-A)

During Nasal lawage in early and late phase, mediators including histamine, Prostaglandin D, Bradekinin and TAME esterase have been demonstrated.

Mast cells are located within the epithelium and submucosa of the upper and lower respiratory tract. Although largest number of mast cells are located within the submucosa, Mucosal type (tryptase only) mast cells migrate through the human Nasal epithelium during natural seasonal grass pollen exposure. Basophils are also found in blown secretions of patient with rhinitis. Thus water soluble allergens are free to interact with IgE sensitized mast cells (and basophils) superficially within nasal epithelium and in nasal secretions. The

resulting rapid release of mediators from cells located in the epithelium or Free on the nasal mucosa are responsible for immediate allergen induced symptoms of itch,. sneeze, watery rhinorrhoea, nasal congestion and blockage. The allergic symptoms are mainly produced by histamine mediator. Histamine is a amine autacoids, present mostly within storage granules of mast cell. Tissue rich in histanine are skin, gastric & Intestine mucosa, liver & placenta. Non most cell histamine occur in brain, epidermis, gastric mucosa & growing region. Histamine is also present in blood, most body secretions, venom & Pathological fluids.

As far examination concern history is very important .The diagnosis is made by combination of clinical history, skin prick test, radioallergo sorbent test, ELISA and nasal mucosa histopathology. Although most of the laboratory tests are good but we will do more stress on nasal Pathology. As far the treatment is concerned, the most important part is to avoid the allergens but it is most difficult. The drugs commonly used are Antihistaminics, Anticholinergics, Mast cell stabilizers, Steroids and Decongestant used topically as well as systemically. The important point in treatment is to find out the etiology and the me of treatment.

Aims & Objects

AIMS & OBJECTIVES

1. "A Histopathological study of the nasal mucosa pre & post Anti allergic Medication including oral & Topical drugs in Allergic Rhinitis in Bundelkhand

2. To compare the Histopathological changes in response to different Anti allergic drugs -

   (i) Oral Drugs : (Older & newer Anti histaminic drugs).

   (a) Older Drugs : Pheniramine, Dimethinedene, Chlorpheniramine

   (b) Newer Drugs: Levocetrizine, Fexofenadina, Ebastine

   (ii) Topical Drugs

   (a) Older Drugs : Xylometazoline, Naphazoline and Hydrocortisone

   (b) Newer Drugs: Beclomethasone, Fluticasone, Budesonide

3. To compare the Histopathological trend in This area with the different National and International studies in Allergic Rhinitis.

4. Comparison will help us to formulate a set line of treatment in Allergic Rhinitis taking into consideration the season, type of presentation and Histopathological Response with clinical response.

Review of Literature

REVIEW OF LITERATURE

Definition:

Von Pirquet (1906) proposed the tenrm allergy, to described a change of reactivity of the living tissues, with increased or decrease sensitiveness due to the formation of specific antibodies. He stated that the vaccinated person behaves in a different manner from him who has not previously been in contact with such an agent, yet he is not insensitive to it. We can only say of him that his power to react has undergone a change.

Jorden (1955) stated "I do not believe that all allergy is an antigen-antibody reactions by any means and I include autonomic dysfunction, psychic changes, endocrine dysfunction etc., under the broad term allergy.

Shambough (1945) states that an allergic individual is one whose sensitivity threshold is so raised to various materials that exposure to small amounts of such material produces in, him an unusual tissue reactions, which is insufficient to cause any such reaction in a normal individual.

Forman sums up the definition very well by stating "Allergy is the sum total of unfavourable stresses and strains which the environment places upon the individual.

Konno et al, 1982 stated that the symptoms of nasal allergy are caused partly by the direct effect of chemical mediators released from mast cells and basophilic cells as result of antigen • antibody reaction and partly by reflex excitation of the efferent nervous pathway resulting from stimulation of sensory nerve endings.

Concept Of Allergy:

a) Allergy as antigen antibody reaction :

Aside from physical allergy, all the conditions cited as examples show the features typical of reaction between antigen and antibody. **Prausnitz and Kustner** in 1923 have shown that injection of serum from patients with asthma hay fever group into normal human skin induced a passive local sensitiztion of the skin of the recepient.

Shambough Jr (1945) states that we do not know that a allergic individual makes him develop antibodies against substance which normally do not stimulate antibody production, except that hereditary is an important factor.

b) Hapten Theory of Allergy :

Wolf- Eisner (1906) suggested that antigenic agents such as drugs might combine with the body proteins to from complex antigen. The specifiety of which was determined by the non antigenic foreign substances, This hypothesis was the basis for the Hapten theory which was fully confirmed some years later by **Landsteiner** (1942) and is now accepted explanation of allergy to drugs and
other non antigenic substances.

c) Enzymatic Concept of Allergy :

According to this theory, the allergic reaction is produced as a result of abnormal enzyme mechanism in the body (**Goldowski,** 1958).

d) Autonomic Dysfunction Theory :

Williams (1952) defined allergy as an inherited predisposition to a localised type of autonomic dysfunction mediated by the cholinergic apparatus of the antonomic system.

e) Acording to white (1992), Although, the precise offending allergen may be difficult to discover, allergic rhinitis by definition is a disease of known aetiology. Certain people produce an abnormal response to various foreign substances. These can be proteins, or else haptens (e.g. Pollens), which combine with amino acids in the body to from proteins. Whereas in non sensitive subject the reticulo-endolhelial system reacts to foreign proteins by producing a specific antibody, susceptible people produce additional reagenic antibodies, associated with the IgE immunoglobulins, and these allergic subjects show a high IgE level in the blood . The Sensitization process is eventually due to the combination of the IgE reagenic antibody with cells such as tissue mast cells. On exposure to the foreign protein the allergen combines with cell- bound reagenic antibodies to release histamine and similar amines and other factors as listed below:

Mast Cell Mediators	
Preformed	**Newly Synthesized**
Histamine	SRS-A
Serotonin	Platelet aggregating factor (PAF)
ECF-A	Prostaglandins
Neutrophil Chemotactic factors	Kallikrein-Kinins
Chymase	Lipid Cbemotactic factors
Heparin	
Antisulfatase A	

Aetiology

(a) Incidence - Vangham (1933) and **Bray** (1957) stated that roughly 10% of general population is frankly allergic and 50% gives history of transient episodes.

Shambaugh (1945) Stated thai at least 90% of chronic nasal infections and 70% of chronic sinus infections can be shown to have an underlying allergic factors responsible for chronicity.

Negus stated that the onset of nasal allergy occurs frequently in first decade of life. In the second and third decades smaller number develop allergic symptoms, and in the fourth, fifth and sixth decades the incidence sharply declines.

The prevalence of allergic rhinitis varies markedly from study to study 0.1% (**Schwartz,** 1952) to 28% (**Malmberg,** 1979).

PREDISPOSING FACTORS

1. **Hereditary :** A positive family history is present in 50% of cases (**Angel James,**1952).

   Urbach (1946) observed, children with bilateral inheritance develop allergy in 45% of cases, those with unilateral inheritance develop allergy amount to 50% and those without a family history, 7-12%.

2. **Constitutional :** Nasal allergy patients have a poor reactive mechanism to environmental stress which is reflected as a autouomic dysfunction.

3. **Infection :** It leads to allergic sensitization to bacterial products and not to cell (Lucos, 1953).

4. **Endocrine : Goldowski** (1958) quoted, adrenal, ovarian and thyroid hormones are capable of making the nasal mucosa more sensitive.

Symptoms may be precipitated by the influence of pregnancy **(Hensel).** About one half of patient of rhinitis in pregnancy are found to have sinus infection **(Sorri, Hantikainen and Kaija, 1980).**

5. **Psychological**

Holmes, Godwell, Wolf, 1950, states many Factors of this nature lead to functional disorder of the nose as a stress phenomenon. This type of predisposition appears to be increasing in frequency and importance as a basic factor.

6. **Physical**

Changes in the humidity and content of the inspired air may perhaps render the nasal mucosa liable to allergic disturbances.

A light stimulus of light, cold, heat or mechanical irritant which would not affect that normal nose may cause nasal symptoms in a sensitive subject.

PRECIPITATING FACTORS

(a) Inhalants

Stafford (1987) States that inhalants form the biggest group and are especially important in adults. A very large number have been recorded and they include dusts, pollens animal emanations, feathers, orris root used in many cosmetic, fungus fumes, wood etc.

(b) Ingestants (Foods)

Are especially important in children. The common food which cause allergy are eggs, meat rice, curds, wheat, milk fried fats.

Pinket (1955) Classified food allergy into two types.

(i) Fixed (5% cases) (ii) cyclic (95% cases)

(c) Infectants :

Stafford 1987, observed liver extracts, insulin and penicillin etc. Can produce allergic reaction.

(d) Chemical Substances:

Drugs like aspirin and potassium iodide can produce hypersensitivity which could be explained by hapten theory of allergy (**Wolf,** 1906)

(e) **Physical Agent:**

Physical agents may precipitate allergic reactions. Exposure to strong sun light, cold heat humidity, barometric pressure changes, wind etc, can precipitate the symptoms in sensitive patients. These probably act as "triggers" and effect the mucosa reflexly as evidence by fits of sneezing which follow stepping out of bed with bare foot on to a cold floor (**Hansel,** 1930).

(g) **Non- Specific Irritants:**

Strong smell, tobacco smoke petrol fumes etc. May directly initiate the hypersensitive mucosa and excite the reaction.

PATHOLOGY

Hiranandani, 1964 described die typical changes in nasal mucosa of a case allergic rhinitis .pathologic changes in nasal mucosa in early stages of allergy is reversible one , so that after the hayfever season the "Water logged" nose return to normal. Long standing allergy, especially when complicated by infection results is irreversible hyperplastic and polypoidal changes in the nasal mucosa.

(a) Local Mucosal Changes

Oedema From intracellular and intercellular transudation of tissue fluid. Marked oedema leads to compression of small superficial vessels and produces the characteristic pale swollen mucosa.

(b) Infiltration with eosinopbils and plasma cells

Eosinophilic infiltration in tunica propria is a characteristic feature of allergic mucosa. Plasma cells are derived from the reticuloendothelial system.

(c) Epithelial Changes

Individual epithlial cells swell as a result of oedema. In perennial allergy, epithelium shows, more hypcrplasia and more degenerations, There is increased number of goblet cells replacing many of the columnar ciliated epithelium.

(d) Excretion of Thin Watery Discharge

This results from increased activity of seromucious glands. Histologically there is hypertrophy of goblet cells and mucosal glands.

(e) Vascular Dilatation

Venous stasis particularly affects the inferior turbinate which become enormously hypertrophied.

(g) Superadded infection

Shambough Jr (1945) stated that at least 70% of chronic sinus infection and 90% of chronic nasal infection have an underlying allergic factor responsible for chronicity. In the mucosa., infiltration by polymorph and lymphocytes predominates. Eosinophils may disappear.

(h) Polyp

Theodure just 1939, reported polypi are more commonly found in conjunction.- with asthma than with nasal allergy.

SYMPTOMS AND SIGNS

Symptoms

Lindqvist et al 1986, states the most characteristic clinical features are sneezing, nasal discharge and nasal obstruction. These symptoms vary in prominence and degree, in different subjects, with period of remission in between.

(I) Nasal Irritation

Rinkel 1962 observed, tickling or itching sensation in the nose is a common symptoms and is usually followed by sneezing. Itching is pathognomonic symptom of allergy.

(II) Sneezing

It is a reflex action produced by stimulation of hypersensitive mucosa and occurs in paroxysmal sudden attacks in most cases. (Councill, 1984)

(III) Nasal Discharge

Lindquvist 1986, states profuse, clear watery nasal discharge associated with sneezing is a prominent symptom. Post nasal discharge is a frequent complaints.

(IV) Nasal Obstruction

The main feature is its alternating character but it may be persistant due to chronic nasal oedema or polypus. (Dale, 1982)

(V) Anosmia

It is some times complained of intermittently or continuously even in the absence of obstruction.

(VI) Headache

A feeling of heavyness or actual frontal headache is a common symptom. Pinkel (1962) stated that irregular occurrence of headache is characteristic of allergy.

(VII) Frequent "Head Colds"

Increased susceptibility to head colds and the tendency of these to last weeks or month instead of days is characteristic feature.

(VIII) Anosmia

It is due to nasal obstruction preventing odours from reaching olfactory area of the nose . It may be intermittent on constant.

(XI) General Symptoms

These are fatigue, lack of concentration and anorexia (Brown, 1979)

(X) Associated symptoms

According to **Mygind & Lewenstein** (1982), manifestations of allergic involvement of other systems are frequently found in cases of allergic rhinitis. Cough and hoarseness of voice are the common laryngeal symptoms. Symptoms of bronchial obstruction results from spasm of bronchial muscle or oedema of mucosa.

Eye symptoms like itching, lacrimation, puffiness of eye lids, redness and oedema of conjunctiva are common especially in cases due to inhalant allergen like pollen. Skin manifestations include itching, urticaria, eczema and skin erruptions recurrent attacks of soreness of pharynx, oedema of uvula, soft palate and lips are seen in some cases.

Gastrointestinal symptoms like distress after food, gastric pain, bloating nausea vomiting, and diarrhoea occur in food allergy. Allergy may

affect the ear, producing deafness, tinnitis or chronic discharge, swollen painful joints, bursitis and myalgia me the allergic symptoms of musculoskeletal system.

Albuminuria, heamaturia, bladder initiative and oedema of prostate due to allergy have been reported (Rinkel.1962)

SIGNS

1. Anterior Rhinosocpy

Mygind (1979) described the various signs of allergic rhinitis. The pale, boggy, bluish tinged mucosa, is characteristic of the well developed allergic rhinitis. Not all allergic individuals exhibit the classical pale, boggy, blue gray mucosa, it may vary from a normal watermelon red to pathologic pale, pinkish white. In many patients with allergy of short duration there is a cheery red colourations described as a cocks comb type of redness.

During an attack there is swelling of the erectile tissue of the turbinals and increased secretions. The mucous membrance, espeically that over inferior turbinates, is often swollen as completely to occlude the passage. If touched with a probe, it is found to be elastic. The application of cocaine produces some retraction and recieves a more complete view, but the improvement is transitory.

In presence of mucoid or purulent cxudate in the vestibule, the atrium, the floor of the nose and the middle meatus is significant. Allergic secretions tend to be more ropy in their consitancy then secretion of inflammatory origin.

2. Posterior Rhinoscopy

Accorrding to Mygind (1979), The clasical pale, boggy mulberry like posterior tips of the inferior turbinates are significant and should suggest the possibility of an allergy. These however may be physiological for certain individuals.

The occurrance of regenerative lymphoid tissue in the tonsillar fossa is indicative of allergy. The hypertrophy of the lateral bands of the pharynx and the posterior pharyngeal wall are highly suggestive of allergy. Pharygeal bands frequently thought to be due to infection alone, have also been proven to be due to allergy.

DIAGNOSIS

1. History

A careful and complete history is the first and next important step in diagnosis. It helps to establish the existance of allergic diseases and guides to detect the specific causative agent.

Shambough Jr. (1945) found that it was more valuable than skin test. A positive family history or occurrence of previous allergic manifestations in the patient are important points to be noted. A search should be made to determine such points on the time of the attacks occur, presence of any seasonal variation, effect of change of environment, presence of any trigger mechanism precipitating the attack, the part played by emotional factors and any special intolerance to article of diet. The symptoms are related to season change of climate, a vacation from work or trip away from home, the allergen is most probably an inhalant. Pollen allergy occurs in lowering seaosns. If patient is better in summer but worse in cold weather or when inside the home or when dust is stirred up, the offending agent is probably

the house dust. Gas formation, distress or headache occurring after eating point to the possibility of a food allergen (Shambough Jr, (1945).

2. X-ray Pam Nasal Sinuses

Charles et al 1977, states X-ray will give information as to the state of nasal sinuses which is otherwise not easily obtained, as transillumination is not informative and is unreliable. There may be mucosal thickening of the antram or other para nasal sinuses. Sometime there may be cyst or cysts over the floor of the antram or fluid level.

3. Antral Puncture

Clear yellow fluid or thick mucus is obtained in uncomplicated cases when there is infection it becomes purulent and bacteriological study is helpful.

4. Nasal Smear

Eosinophilia in nasal mulcous is considerd by an impressive assay of authorities to be pathognomic of an allergic condition. **Gay (1945), Hanis (1951), Hansel. (1953), Glaser (1958) and Jensen (1956). But Ceolding Wood** emphatically states that the functional fate of eosinophil is still unknown and we can accept that eosiniophils occur in nasal tissues after antigen antibody reactions.

Shambough Jr. 1945, stated a Positive Smear may be helfpul in diagnosis but repeated exmination is neccssary to demonstrate them.

A Negative smear does not rule out allergy (Mygind, 1979)

5. Biopsy Of Nasal Mucosa

A tyical structures variation is with infiltration eosinophil, **(Grist Wood, 1982)**. Histopathology from the biopsy piece of inferior turbinate shows papillary proliferation of mucous membrane and hyperplasia of

mucous gland, submucuusa shows the presence of inflammatory cells which is predominatly eosinophil (Bbargava, 1980), epithelium shows changes for single layered columnar epithelium, broadening of basement membrane, and infiltleration with mononuclear and plasma cells are the constant features in HPE (**Hiranandani**, 1964).

TREATMENT

Treatment of allergic rhinitis is far from satisfactory. Ideal treatment should be directerd towards correction of etiological factor, avoidence of allergens and desensitization. However accurate determination of the cause is often difficult. The complex nature of allergy, endocrine dysfunction, and psychological stress make it difficult in many case to apply adequate treatment even when only one of the factor is present. Various methods of treatment have been advocated, each with its own limitations and degree of success.

1. **Avoidance of Precipitating Factor**

   Hagy 1969, quoted this is often successful in case of allergens like articles of food. In others who are sensitive to change of temperature, humidity and direction of wind etc, the precipitation factors cannot be avoided.

2. **Desensitization or hyposensitization**

   This is indicated in most of the cases where the allergen is known but cannot be avoided.

   2.1 Specific hyposentization - The latest theory regarding the mechanism assumes that it stimulates production of an immune or blocking antibody distinct from the reagenic antibody and it blocks the union of allergen and reagin (Hartey.1960).

2.2 Non Specific Hyposensitization - Specific desensitization is successful only with few types of allergens so next attempt was to find out a method which would desensitize or inhibit allergic reactions without the employment of a specific antigen so that it can be applied uniformly to all types of allergy.

3. Antihistaiuines

Dannenberg & Feinberg, (1951), stated these drugs antagonize the action of histamine by a competitive inhibition at receptor site, A Single dose is effective only for a few hours. Development of tolerance after their prolonged use is common.

4. Corticosteroids

Pipkorn (1983) states, we still do not know how these anti inflammatory steroids are useful in allergic rhinitis. It is not substitute therapy because there is no evidence of any adrenal cortical insufficiency in allergic disease. These drugs are not recommended for regular use. Wihl (1984) observed that steoids exert profound undesired metabolic endocrine, neuro-muscular, immuuological and cytological effects. The visual contra-indication of cortisone therapy have to be remembered.

5. **Sodium Cromoglycate**

Wide Et Al (1967), observed its effectively in preventing symptoms, if used before attacks.

6. **Histamine Releasing Agents**

Drugs like compound 48/80 dextran, peptones and stilnamidine on administration cause depletion of histamine in body and a histamine free period ensures during which no allergic reactions can take place (Halpern 1960).

7. **Gamma Globulin**

Histamine binding power of plasma which has its seat in gamma plobulin is found to be reduced in allergic patient. This permits treating the allergic cases with injections of blood serum of gamma globulin of normal person. **Crilland** in 1959 and Bernard Redner in 1963 reported good results with this treatment. The injections probably stimulate synthesis of mobilization of interstitial gamma globulin.

8. **Thyroid Hormone**

In many cases of allergic rhinitis resistant to usual methods of treatment thyroid therapy has been found helpful (Walsh, 1950).

9. **Injections of Sphenopalatinc Ganglion With Alcohol**

This reduces sensitivity; of mucosa and relief of symptom have been reported (Hansel, 1943).

10. **Local Treatment**

(a) Zink Ionization - Proetz (1936) & Bhaugeeva Et Al (1980) stated that, this reduces the permeabiltiy and sensitivity of mucosa to allergens by altering the reaction of fluid bathing the mucosal cells. In perennial allergy it gives relief in 60-80 % cases.

(b) Auto Haemotherapy - Rege and Shah, 1958, tried injections of own blood submucosally in inferior turbinate in cases of allergic rhinitis.

(c) Cauterization Submucosal Diathermy and Cryosurgery - It reduces the bulk of hypertrophied turbinates and relieves nasal obstruction. (Ozenberger 1970 Karjaetal 1975).

(d) Submucosal Injections of Cortico - Steroids- These has been tried in allergic rhinitis and given good results (Gill 1966).

(e) Steroid aerosol - Beclomethasone dipropionate is administered by standard metered aerosol which delivers 50 microgram of drug/puff taken as sniff and has given good results (Brown Storey and Jackson, 1977).

Topical Nasal Drugs

The treatement of allergic rhinitis revolutionized over the last few years by the introduction of various topical preparations of nasal drugs especially topical corticosteroids. Various preparations of nasal drops containing either decongestant and vasoconstriction agent or topical corticcosteroids (Beclomethasone, prednisolone) have been used and have shown good results in the treatment of allergic rhinitis.

Xylometazoline Hvdrochloride - Zee & Dischockm, 1962 tested the effect of xylometazoline nose drops in allergic rhinit's in different ways and claimed it to be effective.

Mesek H 1962 treated various cases of allergic rhinits with xylometazoline nasal drop and described its effectively in allergic rhinitis in his book "clinical experience with xylometazline".

Aschan & Rettner, 1964 explained the various objective, investigations of decongestive effect of xylometazoline in patients of nasal allergy.

Symptomatic relief from nasal congestion associated with allergic rhinitis can be obtained by the use of xylometazoline which exerts its effect by vasoconstriction of mucosal blood vessels which in turn reduces the thickness of nasal mucosa. (Ariens, 1967).

Anggard and Malm, 1983 quoted topical decongestant xylometazoline is more effective than systemic decongestant in the management of allergic rhinitis.

Various' studies have shown in the past that topical nasal drugs preparation containing steroids are the most effective measure in the management of allergic rhinitis.

Proetz (1936' stated that local treatment of allergic rhinitis with zinc ionization gave relife in 60-80% cases of chronic nasal allergy.

Bordley, (1949) reported encouraging results with the hormone used as nose drops in the treatment of chronic nasal allergy,

Goodman Gilman (1950) stales cortisone and ACTH provide symptomatic relief in disease of allergy.

Silicox (1958) reported a study of 174 patients suffering from allergic rhinitis. He treated with topical hydrocrotisone 4 times a day with decongestant phenylephrine hydrochloride. He says that hydrocortisone solution alone is not useful in case of acute rhinitis, but with decongestant it is more effective.

Leopoldkroman and Green, reported 125 patients on whom prednisolone nasal spray was used, 115 obtained significant relief Simon? (1960) reported on 419 patients with allergic rhinitis who were treated with predinisolone injection locally. Benefit was obtained in 78% of his cases.

Hiranandani, 1964 carried out a clinicopathological study of vidian nerve section in the treatment of allergic rhinitis with satisfying results.

Gill, 1966 carried out a clinical and histopathological study of intraturbinate use of steroids in nasal allergy in 104 patients, with success rate of 81.25% of cases.

Halopainen et al, 1971 recognised that in allergic rhinitis topical application of disodium cromoglycate given good results were short lived.

Mygind, 1973 recognised the anplication of topically active steroid was very effective in the treatment of allergic rhinitis.

Brown Storey And Jackson, 1977 administered beclomethasone dipropionate by a standard metered aerosol and gave good results in treatment of allergic rhinitis.

Bhargava et al, 1980 Carried out a clinico histopathological study of local application of silver nitrate in the treatment of allergic rhinitis. 51 cases were selected and nasal biospy was taken before and after treatment. 68.3% of Patients had good relief particularly from sneezing and watering of nose. Potent topical corticosteroids such as intra nasal baclomethasone dipropionate are useful in severe nasal congestion due to allergy.

Cromolyn sodium appears to have some efficacy in suppressing symptoms of allergic rhinitis (**Hendels, Weinberg and Wong,** 1980).

Gale, Soloman & Tao, 1980 carried out a study comprising of the intranasal topical flunisolide therapy in thirty five children with seasonal allergic rhinitis and concluded 64% of the flunisolide treated group noted substantial or total control.

Bharava, Abhyankar, Shah, 1980 treated the 41 patients of allergic rhinits with local application of silver nitrate and found 79.4% patients reported relief significantly.

Petruson 1981 stated xylometazoline (0.1%) nasal drops are effective in the treatment of allergic rhinitis with fewer side effects. He also stated that nasal drop containing ephedrine and /or naphazoline were commonly used but side effects like reactive congestion, tachyphylaxis and rhinitis medicamentosa were ferquently observed.

Malm, Wihl, Lamm and Lindquist, 1981 quoted the value of three objective tests of nasal mucosa in 22 patients with allergic rhinitis treated with a topical cortcosteroid.

Empey, Meddler, 1981 stated that in an acute case of allergic rhinitis, topical decongestant may be the most immediately effective remedy. Their study was based on the use of topical decongestant cantaining pseudoephedrine or phenylepherine in acute cases of allergic rhinitis.

Mygind, 1982 used glucocorticoids topical in allergic rhinitis cases with significant good results.

Swenson, 1982 carried out a study which comprised of topical treatment of allergic rhinitis with a beta adrenocepto:- stimulant (KWD 2131).

Interanasal steroid treatment can reduce methacholine induced nasal secretion, reduce the sensitivity of mucosal irritation receptors and lower the number of basophilic as well as eosinophilic cells in the nasal secretion **(Wihi, 1982).**

Warland 1982 evaluated the effectiveness of a topical steroid flunisolide in thirty four patients with perennial rhinitis. There was a statistically significant difference in favour of flunisolide.

Balle, Pedersen, 1982 quoted that the treatment of allergic rhinitis in a new, halogenated topical areosol packed steroid, Budesonidc had a significantly
better effect.

Hamilton L.H 1982 studied nasal decongestant effect of propylhexidine in patients of allergic rhinitis and found most patients got significant amount of reduction in their symptoms.

Topical cortico steroids administered intranasally are clealry the most effective medications for treatment of chronic allergic rhinitis (Estelle, Simon, 1984).

Asakura Enomoto et al, 1984 examined nasal responsiveness to topical methacholine application in allergic rhinitis and non allergic rhinitis and concluded that methacholine responsinveness was significantly higher in allergic rhinitis.

Wihl 1984 observed the effectiveness of topical intranasal steroid in treatment of allergic rhinitis.

Kwaselow et al, 1985 studied a comparison of intranasal and oral flunisolide in the therapy of allergic rhinitis and evidenced that intra nasal flunisolide is an effective treatment of allergic rhinitis,

Watase, Okuda, 1986 Carried out a study to elucide the effects of different kinds of autonomotropic drugs in the nasal mucosa as well as on the nasal reaction to specific allergens in patients with nasal allergy.

Lindqvist et al, 1986 Recommended the long term safety and efficacy of budesonide nasal aerosol in perennial rhinitis.

Orgel et al, 1986 Observed clinical rhinomanometric and cytologic evaluation of seasonal allergic rhinitis treated with beclomethasone diproprioUate as aqueous nasal spray. They stated topical BDP was rapidly effective in decreasing mean nasal obstruction, rhino-rhea, sneezing and itching symptoms.

Caorado Olliers, 1987 measured the changes in nasal airway resistance following the topical application of histamine by passive anterior hinomanometry.

Welsh et al, 1987 Conducted a randomized clinical trial showing the efficacy of beclomethasone nasal solution flunisolide and cromolyn in relieving symptoms of ragweed allergy.

Topical glucocorticosteroids, singnificantly reduces both the symptoms and level of histamine. TAMP esterase activity and kinin in allergic reactions (**pipkorn et al.** 1987).

Bende and Pipkorn, 1987 highlighted the efficacy of topical levocabastine, a selective H1 antagonist in seasonal allergic rhinitis.

Pipkorn and Everback 1987, designed a study in order to elucidate the interaction in the treatment of allergic rhinitis and the migration of mast cells.

Togias Proud et al, 1987 Aowed the effect of topical tricyclic antihistamines on the response of the nasal mucosa to challange with cold, dry air and histamine.

Patients presenting with typical signs and symptoms of allergic rhinits may respond to avoidance of allergens and to medications for symptomatic relief (**Safford** 1987).

Topical synthetic corticosteroids and orally administered delta- 1 steroids are the commonly prescribed medications used alone or in various combination in allergic rhinitis (**Berman,**1988).

Naclerio, 1988 observed that pretreatment the patient of allergic rhinitis with topical steroid flunisolide several days before the pollen season reduces the early response of allergen and inflammation associated with chronic allergic rhinitis.

Meltzer, 1988 documented that topical Corticosteroids including flunisolide therapy decreases the symptoms, improves patency of nasal airways in patients with allergic rhinitis.

Topical nasal decongestants give fast relief from nasal congestion, but their over use may result in rebound congestion (**Burse**, 1988),

Bunnag et al 1988 suggests that intranasal budesonide is an effective and well tolerated treatment for perennial rhinitis.

Pechler et al, 1988 Carried out a clinical comparison of systemic methyl prednisolone acetate (MPA) verus topical budesonide in patients with seasonal allergic rhinitis and showed the efficacy of topical budesonide over MPA in allergic rhinitis,

Ballas Seltzer et al, 1990 evaluated symptoms reflief, nasal airflow, nasal cytology and acceptability of two formulations of flunisolide nasal spray in patients with perennial allergic rhinitis.

The use of decor gestants (sympthomimetics) is limited by the so called rhinopathia medicamentosa, when the necessary treatment exceeds 2 or 4 weeks (**Albergger, K.** 1990).

Klemeaston Lindquist et al, 1990 performed a study showing the effect of single dose of topical glucocorticoid and a cycloxygenase inhibitors on allergen induced changes in nasal reactivity with good results.

Bonsquest and Michel, 1990 reviewed the therapeutic approach to seasonal allergic rhinitis and observed that

(1) Topical vasoconstrictors are effective but cause side effects when treatment is prolonged.
(2) Topical corticosteroids are highly effective and safe.

Naclerio, 1990 demonstrated that histaminic plays an important role in the mediation of allergic rhinitis.

Darnell, Pecard and Recbard, 1994 carried out a double blind comparison of fluticasone propionate aquous nasal spiny, Terfenadine tablets and placebo in treatment of patients with seasonal allergic rhinitis to grass pollen and showed higher efficacy of fluticasone topically.

Nonsteroidal anti inflammatory drugs have weak effects in allergic rhinitis as compared with glucocorticoids (**Malm,** 1994).

Kobayashi, 1994 stated that nasal steroids can be used and are safe and effective as antihistamine in controlling symptoms of allergic rhinitis.

Moper, 1995 Reviewed the therapeutic use of pregnendiones in allergic rhinitis.

Lemanske et al, 1990 observed the significance ana potency of topical fluticasone propionate in allerigic rhinitis.

Treatment with mtranasal flimisolide in allergic rhinitis induces marked improvement of clinical symptoms and reduces the total IgE in

nasal secretion. (**Pagenelli et al,** 1991). topical use of fluni.solidc in the treatment of perennial rhinitis induces marked improvement of clinical symptoms and that it exerts its effects through its antinflammatory action on nasal mucosa.

Anrihistamine alone cannot control all of symptoms of allergic rhinitis. However, the combination ot antihistamine with topical corticosteroids is very effective (White and Kaliner 1992).

Meltzer, Orgel et al, 1992 studied the topical acitivity of ipratropium bromide and anticholinergic agent in perennial allergic rhinitis and observed its effecinveses in decreasing the rhinorrhoea in patients suffering from allergic rhinitis.

Pedersen, Mygind et al, 1991 observed that budesonide delivered as pure powder is effective and safe for the treatment of seasonal allergic rhinitis.

Macrophages are involved in inflammatory processes of allergic rhinitis (**PipKorn et al,** 1991).

Welch, Garcia et al, 1991 had shown the high effectrveness of topical triamcinolone in allergic rhinitis cases.

Heyning & Rossel, 1991 stated that topical application of levocabastine a potent H1 antagonist yielded good clinical results in allergic rhinitis.

Dechant & Goa, 1991 stated that Levocabastine nasal spray is better than sodium cromoglycate and placebo in the allergic rhinitis.

Scadding Lund et al., 1991 shown the clinical and physiological effects of fluticasone proprionate aqueous nasal spray in the treatment of perennial rhinitis with good results.

Paganeli et al, 1992 stated that

Ratner et al, 1992 stated that fluticasone proprionate given once daily is as effective for seasonal allergic rhinitis as beclomethasone dipropionate given twice daily.

Bryson and Faulds; 1992 reviewed the therapeutic potential of intranasal fluticasone propionate in allergic rhinitis.

Sim Hilsmeir et al, 1992 studied the effect of topical corticosteroid in various patients of allergic rhinitis and proved its efficacy.

Swensson & Pipkorn, 1992 stated that topical vasoconstrictor oxymtazoliue does not affect hislamine Induced mucosal exudation of plasma in human nasal airway.

Topical glucocorticoids inhibit allergen induced activation ofeosinophil in allergic rhinitis (**Lozewic et al,** i992).

Krause, 1992 stated that antihistaminics are the mainstay treatment of allergic rhinitis and lopiuil Jccongcsuuit should be added for a short time to prevent rebound.

Meltzer, 1992 stated that topical anticholinergic medication ipratropium bromide was safe and effective in reducing the troublesome symptoms of allergic rhinitis.

Bunnag, Jore and Wong, I992 Proved the efficacy of topical budesonide and oral astemizole in allergic rhinitis.

Long term use of topical coritosteroid in the nose is not harmful to the nasal mucosa. (**Bende and Mark,** 1992).

Mabsyl, 1993 studied the topical pharmacotherapy for allergic rhinitis and stated that it prevents or ablates both the acute and late phase of allergic response.

Birchall Henderson et al, 1994 studied the effect of topical sodium cromoglycate as intranasal his famine challenge in allergic rhinitis and observed its potent antinflammatory effect.

Thus we can expect better results in allergic rhinitis with topical corticosteroid. Newer topical drugs has proven to be more effective than older topical drugs, in the present study. The topical effect of nasal drugs in allergic reunite, however, need to be studied further.

Material and Methods

MATERIAL & METHOD

The present study **"A Histopathological study of nasal mucosa Pre and post antiallergic medication including oral and topical drugs in Allergic Rhinitis in E.N.T. department of M.L.B. Medical College and Hospital Jhansi."**

A majority of patients belonged to Jhansi district and nearby areas. Patients not willing to attend hospital regularly for treatment and follow up were not included. Total Number of Cases studied were 68.

Diagosis of allergic rhinitis was made mainly on clinical history and nasal examination of findings. A Special Case Sheet Showing all the details to be gathered, was made and finding recorded.

HISTORY

A detailed history was taken of all patients as Follows –

1. Complaints

- Nasal Obstruction - Whole day, Morning or Evening
- Nasal discharge - Watery, Mucoid or Mucopurulent
- Sneezing - No. of attack, variation according to time, Precipitteting factor
- Sense of Smell -
- Headache - Unilateral, Bilateral, Frontal, Occipetal or Generalised
- Itching - Nose, throat, Eye or Palat
- Swelling - Face, Eye

2. Attacks

With particular attention towards the duration, frequency, seasonal variation and excitants for the attacks.

(i) **Other allergeis :** History of Asthma or allergy was taken.

(ii) **Discharge :** The nature and quantity of nasal discharge.

(iii) **Past illness :** History of any illness in the past whether allergic or non allergic and if any surgical intervention was required.

(vi) **Drug / Food allergy :** History regarding any allergy to milk, aspirin, sulphas, pollen cosmetics etc. was asked for.

(v) **Family History :** A Family history of allergic rhinitis was asked for .

CLINICAL EXAMINATION

A Thorough clinical examination was done to rule out any infective or obstructive cause for this symptomatology and for any nasal pathology like nasal deviation and polyposis etc.

1. Nose

(a) External Appearance

(b) Anterior rbinoscopy

- Vestibule
- Mucous mimbrane
- Turbbates-size and color
- Secretion - if any the nature and amount.
- Meptie were examined for the presence of any discharge.
- Septum and airway if adequate

(c) Posterior rhinoscopy

- Patency of Eustachian tubes

- Inspection of posterior ends of the inferior and middle turbmate .
- presence of any discharge

2. Throat

- Orodental hygiene, tongue, soft and hard palate .
- Pillars of tonsils, tonsils, post pharyngeal wall.
- Indirect Laryngoscopy

3. Ears

- Pinna
- External auditory canal
- Tympanic membrane
- Hearing

4. General Examination

A Routine general physical examination was done

INVESTIGATION

Blood Examination :

The haemoglobin percentage, total leucocyte count and the differential leucocyte count was done for all patients. DLC was done to look for eosinophilia.

X-ray Paranasal Sinuses:

An x-ray PNS occipitomental view was done in each case to rule out any associated sinus infection In the patients who were having maxillary sinusitis, an antral wash out was done they were not included in die study.

Inferior Turbinate Boipsy :

Once a clinical diagnosis of allergic rhinits was made each patient was subjected to biopsy of the mucous msmbrane of inferior turbinate. The side from which the biopsy was to be taken was first 2% xylocain with adrenaline was applied with cotton and then injected 2% xylocain with adrenaline, then wait for 5 minute and then the help of microcup forcep and microscisser a small peice of tissue was taken from patient inferior turbinate. The tissue was stored in 10% formal saline and sent for histopathological examination. The biopsy was repeated after 3/4 week of Anti allergic, (older & Newer groups oral and topical spray) drugs in each patients again and biopsy specimen again subjected to histopathological examination. When making the second biopsy from anather inferior turbinate.

Histotogical Technique

After fixation in formal saline for 24 hours the tissue was processed by autotechnicon and embedded in paraffin and blocks were prepared. Sections were cut in weswox opte microtome at 4 micron and routine haematoxyline and eosin (H&E) staining was done.

Haematoxylin and Eosin Staining Process:

- Bring sections to water
- Stain in Ehrlich's hematum - Till Overstrained (10-15min).
- Rinse in water
- Differentiate in acid alcohol (1% conc. HCL in absclute-methylated sprit) till only nuclei are stained.
- Rinse in running water for 5 min
- Differentiate eosin in running water.
- Dehydrate in alcohol, clean and mount it.

Result

The cells take purple stain, the rest of all tissue takes pink stain.

A Special staining of Mast Cell (Hughesdons Metachromatic Method)

(Hughesdon, P.E. (1949)J. Roy, Micr, Soc., 69.1.)

Mast cells are found in tissues under a variety of conditions both normal and pathological.

This Azur-uranyl nitrate metachromatic method of Hughesdon's in excellent for mast cell granules.

Required

1. 1 per cent aqueous potassium permanganate.
2. 5 per cent aqueous oxalic acid.
3. Either one of these three Azur stains can be used :

   1 per cent aqueous Azur A.

   0.2 per cent aqueous Azur B.

   1 per cent aquesous Azur C.
4. 0.5 per cent aqueous uranyl nitrate.

MAST CELL GRANULES

To Stain :

1. Bring sections to distilled water.
2. Oxidise with the potassium permanganate solution one to three minutes.
3. Wash in tap water
4. Treat with oxalic acid solution until colourless.
5. Rinse in distilled water followed by tap water.
6. Stain with Azur A, B or C for two minutes.
7. Rinse off stain with uranyl nitrate solution and replace with fresh, gently rocking the slide to ensure even differentiation. The time

varies between ten to sixty seconds. When the section is pale blue, rinse in tap water.

8. Blot.
9. Quick rinse in absolute alcohol or 74 O.P. spirit.
10. Clear in Xylene.
11. Mount in balsam or D.P.X.

Result :

Mast cell granuless ..... crimson orred other tissue elements, including muscle striation ..... various shades of blue.

Treatment

After making diagnosis full details about nasal biopsy (Before and after treatment) and also the alternative modes of treatment available were explained to the patient. Only those patients willing for this treatment ad also willing for close follow up were selected for the study.

Three topical nasal drugs chosen for comparative clinical as well as histopathological study.

These drugs were -

(1) Older group of antiallergic - (a) Oral antihistaminic – Phemiramine, Dimethinedene, Chlorpheniramine

(b) Topical - Xylometazoline, Naphazoline and Hydrocortisone

(2) Newer group of antihistaminic – (a) Levocetrizine, Fexofenadine, Ebastine

(b) Beclomethasone, Fluticasone, Budesonide

Xylometazoline, Naphazoline (Decongestant) and Hydrocortisone

Decongestant when applied topically to nasal mucosa in from of either nasal drops or aerosol, acts as a decongestant in allergic rhinitis.

It is a sympathomimetic amine of the imidazoline class. It is direct agonist al α_2 adrenoceptors and has no action on β adrenoceptors. It produces a rapid and prolonged vasocostriction lasting for upto 8-12 hours.

Hydrocortisone is a short acting steroid. It acts or for short time it marketed with Naphazoline under the trade name of efcorlin. Hydrocortisone part is having some systemic side effects.

MODE OF USE

Dosage - adults including children over age of 12 years. 2-3 drops of either 0.05% or 0.1% solution in each nostril repeated at intervals of 8-12 hours maximum for 3 weeks. Xylometazoline in marketed under the trade names of otrivin (Ciba-Geigy), Decon (cadila). Xylmctazoline nasal drops contain 0.1% arid pediatric nasal drop contain 0.05%.

Newer Topical Drugs

Budesonide, Beclomethasone, Fluticasone

Topical corticosteroids have been widely used for years in the treatment of allergic rhinitis. Budesonide is a new non halogenated corricosteroid for topical use in cases of allergic rhinits. Fluticasone is a synthetic trifluorinated cortisteroid and Beclomethasone also a steroid. These steroids with a high ratio of topical to systemic effect has recently been synthesized (Thalen & Brattsand, 1979), Earlier clinical studies have demonsirated a favorable effect on allergic rhinitis.

Budesonide in marketed under the trade name of Rhinocort, Baclomethasone is marketed under the trade name Econase and Fluticasone is marketed under the trade name Econase

Doses :

Fluticasone	- 200mcg/day	50mcg/spray
Beclomethasone	- 400mcg/day	50 mcg/spray
Budesonide	- 200mcg/day	50 mcg/spray

At each visit of the patients, under going therapy of with one of the above mentioned topical nasal drug, were asked whether they had noticed any adverse effects.

APPENDIX

(A) CASE RECORD

S. No. : _____ Date : _____

Name : _____ Age / Sex _____

Registration No.: _____

COMPLAINTS :

1. Nasal Obstruction
2. Nasal Discharge
3. Sneezing
4. Other complaints, if any

HISTORY OF PRESENT ILLNESS :

PAST HISTORY :

IDIOSYNCRASIES :

FAMILY HISTORY :

PERSONAL HISTORY :

TREATMENT HISTORY :

GENERAL PHYSICAL EXAMINATION :

ENT EXAMINATION :

Nose :

1. External Appearance
2. Anterior Rhinoscopy
   - Vestibule

- Septum
- Nostril
- Airway
- Tubinates (Size and Colour)
- Discharge (Character and Amount)
- Meatus (Middle and Inferior)

3. Posterior Rhinoscopy

Throat

- Orodental Hygine
- Oral Cavity
- Oro Pharynx
- Laryngo Pharynx

Ears

SYSTEMIC EXAMINATION :

LABORATORY INVESTIGATIONS :

- Blood Hb%, TLC, DLC
- Urine RE
- Nasal Smear
- X-ray PNS
- Nasal Punch Biopsy

TREATMENT GIVEN :

FOLLOW UP :

Photograph - I
Showing Inferior turbinate biopsy procedure

Photograph - II
Showing Instruments used in Inferior turbinate biopsy procedure

Photograph - III
Showing Nasal drugs used in Allergic rhinitis

Observations

OBSERVATIONS

The present study was carried out on 68 patient who were suffering from allergic rhinitis attending the out patient department of E.N.T., M.L.B., Medical College, Jhansi, from July 2003 to September 2004.

The patients were selected from both sexes and were in the ages varying from 13-53 years.

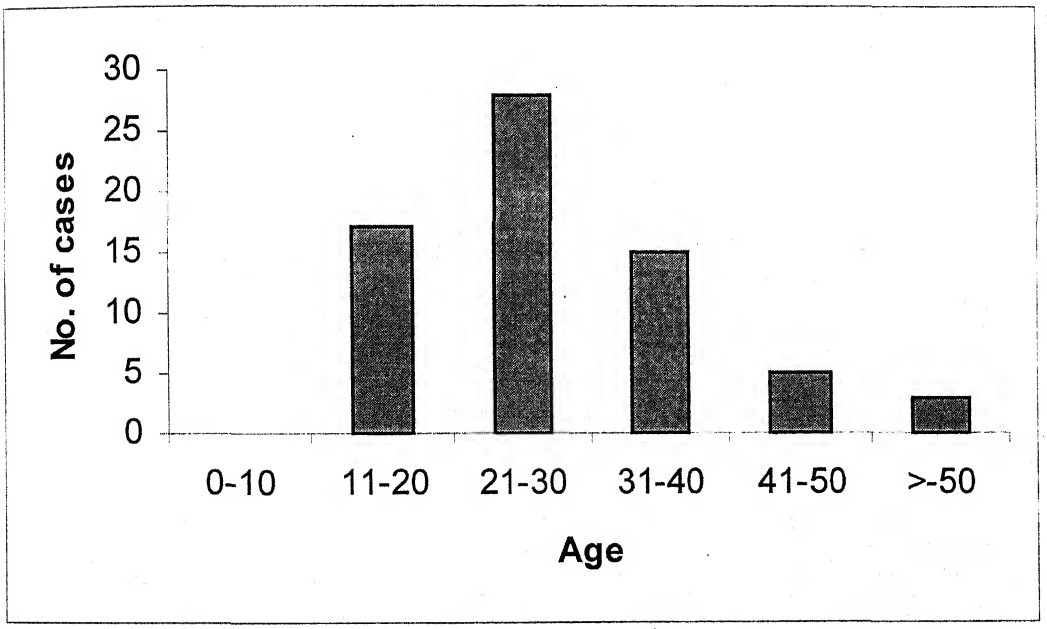
1. Age Distribution

TABLE –I
SHOWING AGE DISTRIBUTION AMONGST 68 CASES OF ALLERGIC RHINITIS

S.No.	Age	No. of Case	Percentage
1.	0 – 10	0	0%
2.	11 – 20	17	25%
3.	21 – 30	28	41.18%
4.	31 - 40	15	22.05%
5.	41 - 50	05	7.35%
6.	> - 50	03	4.41%

Table – 1 shows the age distribution in patients suffering from allergic rhinitis. Majority of patients between the age group of 11-30. (66.18%). The youngest patient was 13 years and the oldest patient was 53 year old.

Table - I



2. Sex Distribution

TABLE – II

SHOWING SEX DISTRIBUTION AMONGST 68 CASES OF ALLERGIC RHINITIS

S.No.	Sex	No. of Cases	Percentage
1	Male	44	64.70%
2	Female	24	35.30
Total		**68**	**100%**

Table – 2 shows the sex distribution in patients suffering from allergic rhinitis. Majority of patients i.e. 44 (64.70%) were males.

3. Type of Allergy Rhinitis

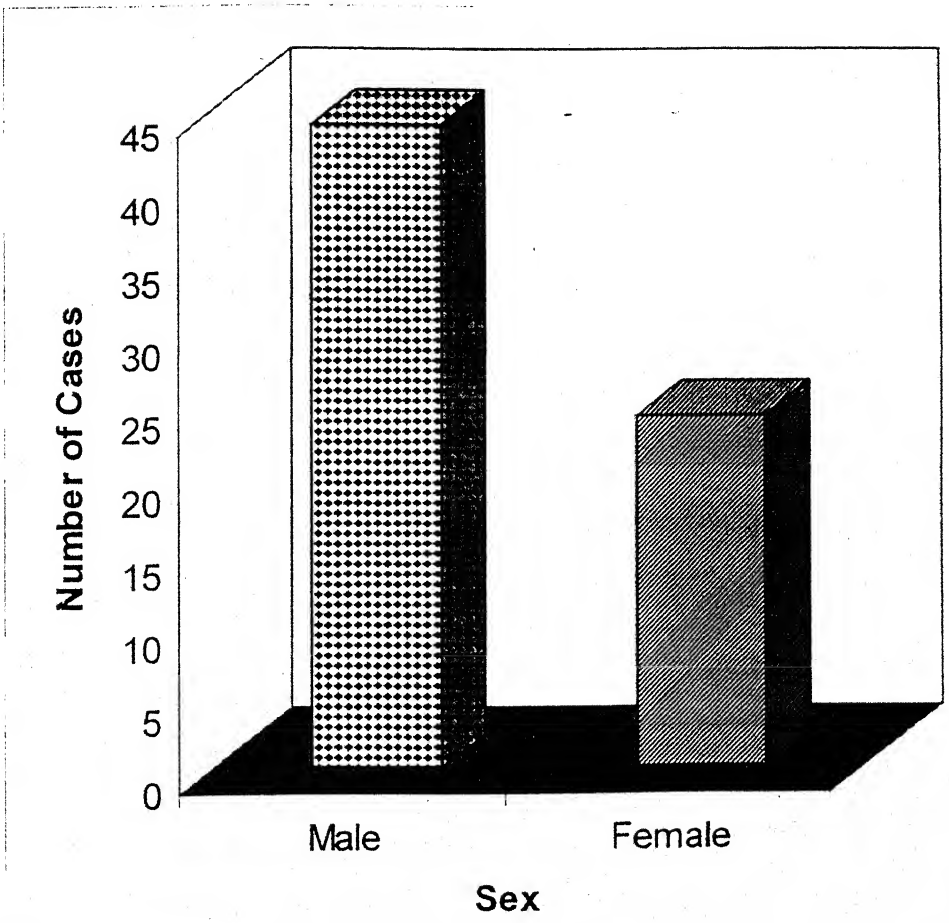
TABLE – III

SHOWING CLASSIFICATION OF ALLERGIC RHINITIS AMONGST 68 CASES

S.No.	Type of Allergy	No. of Cases	Percentage
1.	**Seasonal** (i) Winter (ii) Summer (iii) Rainy	**20** 12 06 02	**29.41%**
2.	**Perennial**	**34**	**50%**
3.	**Mixed**	**14**	**20.59%**
Total		**68**	**100%**

Table – 3 shows the most common cause of allergic rhinitis is perennial rhinitis.

Table - II



4. Occupational Incidence

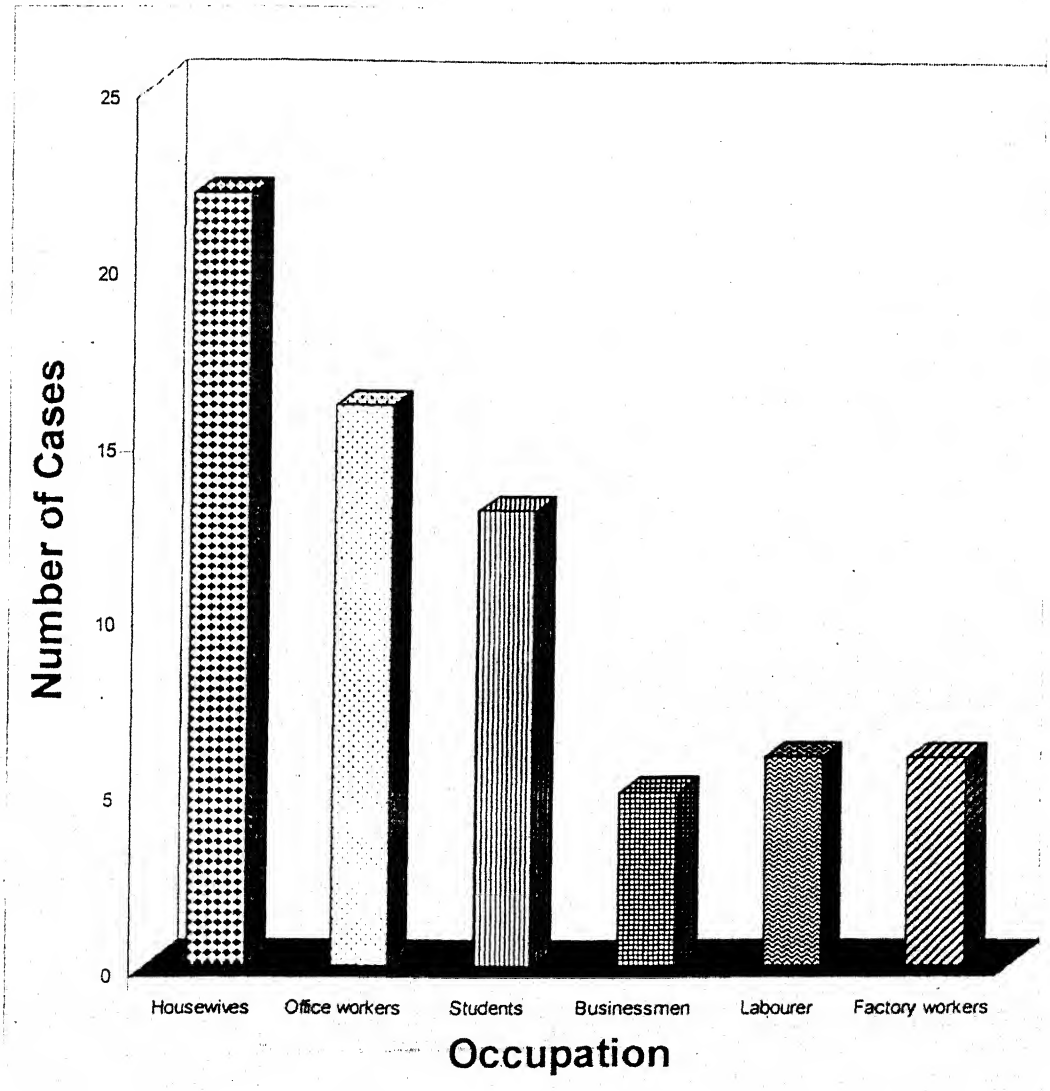
TABLE – IV

OCCUPATIONAL INCIDENCE AMONGST 68 CASES OF ALLERGIC RHINITIS

S.No.	Occupation	No. of Cases	Percentage
1	House wives	22	32.35%
2	Office workers	16	23.53%
3	Students	13	19.12%
4	Businessmen	5	7.35%
5	Labourer	6	8.82%
6	Factory worker	6	8.82%
Total		**68**	**100%**

Table – 4 shows the incidence of allergic rhinitis in patients in relation to the occupation. Incidence was found to be high among house wives and office workers.

Table - IV


25
20
15
10
5
0
Number of Cases
Housewives
Office workers
Students
Businessmen
Labourer
Factory workers
Occupation

5. Causative Factors

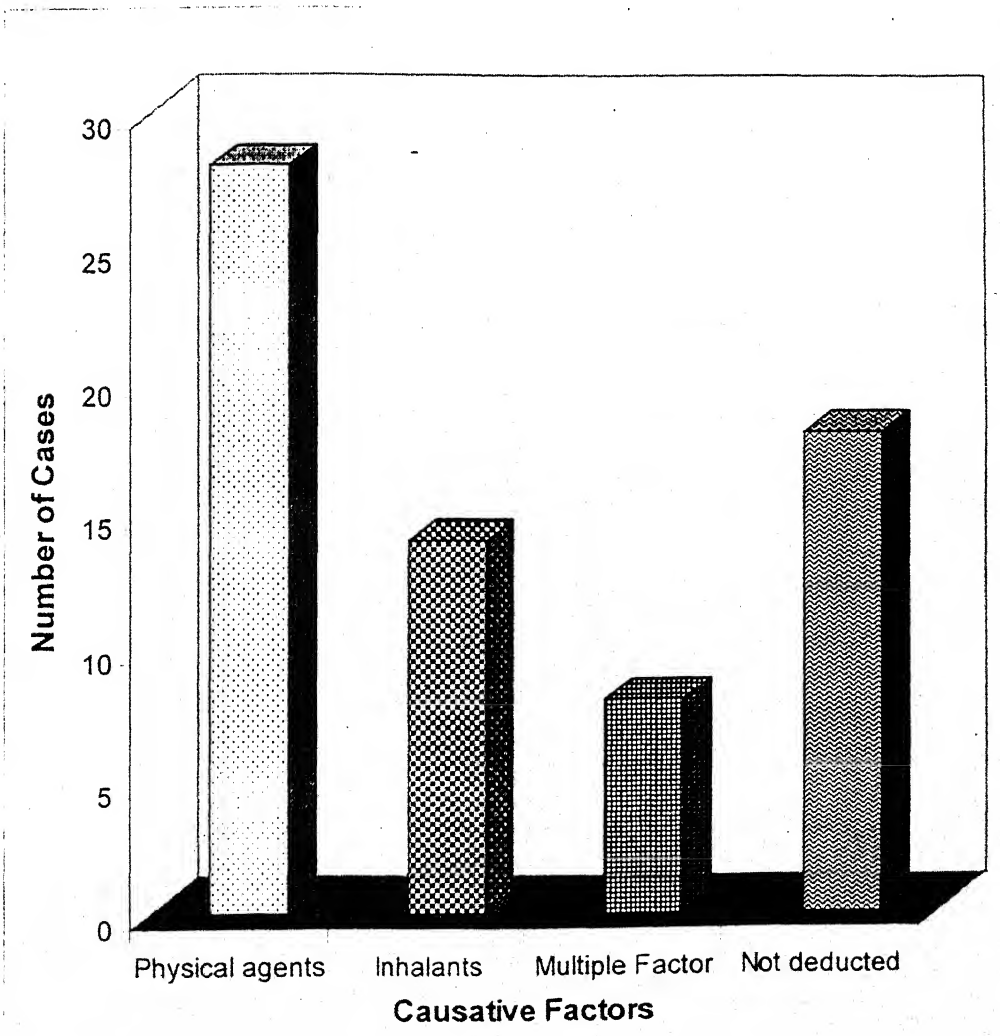
TABLE – V

SHOWING CAUSATIVE FACTORS AMONGS 68 CASES OF ALLERGIC RHINITIS

Causative factors	No. of Cases	Percentage
A. Physical agents	**28**	**41.17%**
Change in Temperature		
Cold Drinks		
Cold bath		
Exposure to cold		
Smokes and fumes		
B. Inhalants	**14**	**20.58%**
House dust and dusty winds Old damp houses and Leathery goods		
C. Multiple Factors	**8**	**11.76%**
Food	6	
Cosmetics	1	
Drug and other	1	
D. Not detected	**18**	**26.47%**
Total	**68**	**100%**

41.17% cases were found to have allergy to physical factors. Most of these were sensitive to change in atmospheric temperature. 20.58% were found allergic to inhalants. Most of them had their attack on exposure to house dust and dusty wides. 11.76% cases had multiple sensitivity. In 26.47% cases no specific causative factor be determined.

Table - V


30
25
20
15
10
5
0
Number of Cases
Physical agents
Inhalants
Multiple Factor
Not deducted
Causative Factors

6. Family history

TABLE – VI

SHOWING FAMILY HISTORY OF ALLERGIC DISEASES AMONGST 68 CASES OF ALLERGIC RHINITIS

S.No.	Family History	No. of Cases	Percentage
1	Positive	28	41.17%
2	Negative	40	58.83%
Total		**68**	**100%**

The history of allergic diseases in the family was positive in 28 (41.17) cases.

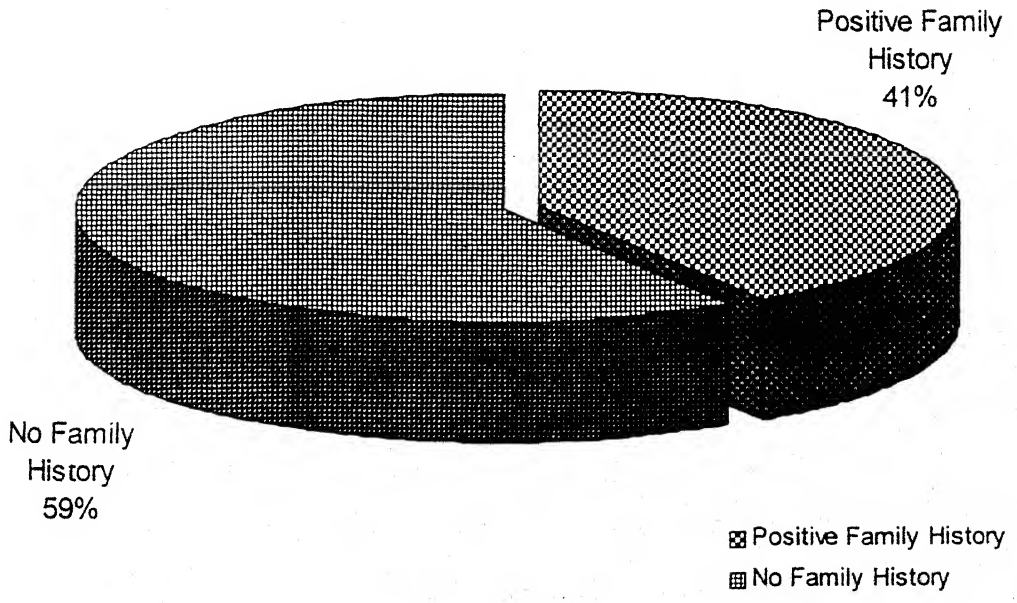
7. Duration of Symptoms

TABLE – VII
DURATION OF ILLNESS AMONGST 68 CASES OF ALLERGIC RHINITIS

S.No.	Duration in Years	No. of Cases	Percentage
1	0-2	24	35.28%
2	2-4	16	23.54%
3	4-6	15	22.06%
4	6-8	5	7.35%
5	Above 8	8	11.76%
Total		**68**	**100%**

Table – 7 shows the duration of symptoms in year and number of patients in each group. The duration varied from 3 months to 17 years.

Table - VI


Positive Family History 41%
No Family History 59%
Positive Family History
No Family History

8. Personal History

TABLE – VIII

SHOWING PERSONAL HISTORY AMONGST 68 CASES OF ALLERGIC RHINITIS

S.No.	Personal History	No. of Cases	Percentage
1	Smoking	12	29.41%
2	Alcohol intake	8	11.77%
3	Coffee & Tea	48	58.82%
Total		**68**	**100%**

12 cases used to smoke 8 cases used to drink and most of them used to take coffee and tea.

9. Psychosomatic History

Most of them were chronic cases and some what anxious and depressed because of protracted illness but three cases were depressed much.

10. Presenting Features

TABLE – IX

SHOWING VARIOUS SYMPTOMS AMONGST 68 CASES OF ALLERGIC RHINITIS

S.No.	Symptoms	No. of Cases	Percentage
1	Sneezing	64	94.17%
2	Nasal Obstruction	51	75.00%
3	Rhinorrhoea	44	64.70%
Total			**100%**

Table – X shows the various symptoms of patients of allergic rhinitis.

(a) **Sneezing :** In our series all expect four cases complained of sneezing frequently

(b) **Nature of Discharge :** 44 cases complained of nasal discharge and in most of the cases, discharge was profuse and watery.

(c) **Nasal Obstruction**

TABLE – X
SHOWING NATURE OF DISCHARGE AMONGST 68 CASES OF ALLERGIC RHINITIS

S.No.	Nature of Discharge	No. of Cases	Percentage
1	Watery profuse	22	50.00%
2	Watery scanty	20	45.45%
3	Mucoid	02	4.55%
Total		**44**	**100%**

TABLE – XI
SHOWING DEGREE OF NASAL OBSTRUCTION AMONGST 58 CASES OF ALLERGIC RHINITIS

S.No.	Degree of obstruction	No. of Cases	Percentage
1	Unilateral	8	15.68%
2	Bilateral	20	29.4%
3	Cyclically Bilateral	23	25.09%
4	No obstruction	17	25%
Total		**68**	**100%**

51 cases complained of nasal obstruction (Table-XII) Cyclically Bilateral nasal obstruction was a predominant symptom next to sneezing.

10. Associated Symptoms

TABLE – XII
SHOWING OTHER ASOCIATED SYMPTOMS AMONGST 68 CASES OF ALLERGIC RHINITIS

S.No.	Symptoms	No. of Cases	Percentage
1	Rec. Attacks of common cold	45	66.17%
2	Recurrent headache	34	50.00%
3	Itching nose	21	30.88%
4	Post nasal discharge	3	4.41%
5	Change of olfaction	16	23.53%
6	Throat symptoms and cough	26	38.24%
7	Inching eyes & Lacrimation	8	11.76%
8	G.I. Symptoms	-	-

Table shows XIII in the majority of cases most prominent associated symptoms were recurrent attacks of common cold, itching of nose, eyes, ears and irregular dull frontal headache.

Sings : - (Anterior Rhinoscopy)

(a) Colour nasal Mucosa

TABLE – XIII

COLOUR OF NASAL MUCOSA AMONGST 68 CASES OF ALLERGIC RHINITIS

S.No.	Colour	No. of Cases	Percentage
1	Pale Bluish	52	76.47%
2	Pink	16	23.53%
	Total	**68**	**100%**

Table – XIV shows the colour of nasal mucosa in patients of allergic rhinitis, most of the patients the 52 (76.47%) had Pale Bluish coloured mucosa the remaining 16 patients (23.53%) had a pink of normal coloured mucosa.

(b) Size of Inferior turbinates

TABLE – XIV

SHOWING DEGREE OF TURBINATE ENLARGEMENT AMONGST 68 CASES OF ALLERGIC RHINITIS

S.No.	Colour	No. of Cases	Percentage
1	Gross enlargement	12	17.65%
2	Moderate enlargement	17	25.00%
3.	Mild enlargement	35	51.47%
4	No enlargement	4	5.88%
	Total	**68**	**100%**

Table – XV shows the different degree of enlargement of the inferior turbinate in patients of allergic rhinitis. Out of 68 cases 12 patients (17.65%) had complete blockage of nasal cavity by the inferior turbinate, 17 patients (25.00%) had Moderate enlargement of the turbinate 35 patients (51.47%) had mild enlargement of the inferior turbinate, four patient had no enlargement.

(c) **Nasal Discharge :** Nasal discharge varied in consistency from watery to mucoid in nature (Table -IX)

INVESTIGATIONS

(a) Hb% TLC, DLC

This was done as a routine in all cases, DLC was done to see eosinophil count.

TABLE – XV

SHOWING EOSINOPHIL COUNT AMONGST 68 CASES OF ALLERGIC RHINITIS

S.No.	Eosinophil count	No. of Cases	Percentage
1	Eosinophil count (0-6%)	14	20.59%
2	Raised eosinophil count (>6-10%)	20	29.41%
3	Raised eosinophil count (> 10%)	34	69.38%
	Total	**68**	**100%**

34 patients (50%) suffering from allergic rhinitis had a raised (>10%) eosinophil count where as 20 patients (29.41) had a eosinophil count between 6-10%.

11. Biopsy of Mucusa of inferior turbinates

In all cases a pre-treatment and post treatment biopsy was done. The histopathological picture was compared in each case.

The patients were followed up for as long as possible in the short duration of this study. In spite of best persuasion only 61 cases (89.70%) returned for follow up, few of them were even contracted by post, but they did not turn up. The follow up study was made on 61 cases only. These patients returned for re-examination after a variable duration of completion of therapy for follow up for several times after therapy.

TABLE – XVII
SHOWING DURATION OF FOLLOW UP AMONGST 61 CASES OF ALLERGIC RHINITIS

1	Between 1-2	12	19.67
2	Above 2-4	17	27.87
3	Above 4-6	28	45.90
4	Above 6-8	4	6.56
Total		61	100%

Table XVII shows that out of 61 patients large number of cases 45.90% came for follow up between 4-6 weeks, 27.87% cases returned between 2-4 weeks, 19.67% between 1-2 weeks, and 6.56% between 6-8 weeks after therapy.

RESULT

(a) **Symptoms**

Table 23 and 24 show the degree of relief in patients of allergic rhinitis treated with (i) Older drug, (a) Oral antihistaminic– Phemiramine, Dimethindene, Chlorpheniramine (b) Topical drugs – Xylometazoline, naphazoline and Hydrocortisone, (ii) Newer drug (a) Oral antihistaminic – Levocetrizine, Fexofenadine, Ebastine, (b) Topical drugs – Beclomethasone, Fluticasone. Budesonide.

Table – XVIII
SHOWING DEGREE OF RELIEF FOLLOWING OLDER DRUG THERAPY AMONG 30 CASES OF ALLERGIC RHINITIS

S.No.	Symptoms	No. of Cases	Complete response	Fair response	Poor response
1.	Sneezing	28 (93.34%)	10 (35.71%)	11 (39.29%)	7 (25%)
2.	Nasal Obstruction	20 (66.66%)	8 (40%)	9 (45%)	3 (15%)
3.	Rhionorrhoea	22 (73.34%)	7 (31.82%)	10 (45.45%)	5 (22.73%)

Table – XIX
SHOWING DEGREE OF RELIEF FOLLOWING NEWER DRUG THERAPY AMONGST 31 CASES OF ALLERGIC RHINITIS

S.No.	Symptoms	No. of Cases	Complete response	Fair response	Poor response
1.	Sneezing	30 (96.77%)	24 (80%)	05 (16.67%)	01 (3.34%)
2.	Nasal Obstruction	21 (67.74%)	18 (85.71%)	03 (14.28%)	-
3.	Rhionorrhoea	20 (64.51%)	14 (70%)	05 (25%)	01 (5%)

The post therapy symptomatology was kept in three categories, in which there was complete absence of symptom considered as good, relief in the symptoms as fair and no improvement as poor. Sneezing, nasal obstruction and rhinorrhoea markedly reduced in majority of cases, 5-7 cases with older group of antihistaminic and one with newer group of antihistaminic noticed no relief in the symptoms.

(b) Histopathology

Results of histopathology were based upon the reduction in cellular infiltration, few mucous and serous glands no evidence of prominent dilated duct, decrease or absence of eosinophil infiltration, reduction in stromal oedema.

Table – XX
SHOWING RESULTS IN POST THERAPY HISTOPATHOLOGY WITH OLDER GROUP OF DRUGS

S.No.	No. of casses	Results			
		Complete response	Fair response	Poor response	No response
1	30	9	10	6	5
2	Percentage	30%	33.33%	20%	16.67%

Table – XXI
SHOWING RESULTS IN POST THERAPY HISTOPATHOLOGY WITH NEWER GROUP OF DRUGS

S.No.	No. of casses	Results			
		Complete response	Fair response	Poor response	No response
1	31	22	6	2	1
2	Percentage	70.97%	19.35%	6.45%	3.23%

Table XX - XXI show that the results in histopathology after thearpy were better newer group of drug.

Table – XXII
SHOWING HISTOPATHOLOGY CHANGE POST THERAPY

S. No.	Structure	No. Patients	Mild	Moderate	Severe
1	Epithelium transitional	58	-	-	-
2	Eosinophil infiltration	44	4	30	10
3	Edema	48	28	12	8
4	Lympocyte infiltration	45	8	28	9
5	Plasma Cell infiltration	18	12	5	1
6	Mast Cell infiltration	16	10	6	
7	Squamous Metaplasia	3	3	-	-
8	Fibrosis	8	6	2	-

1. Eosinophil = Mild Degree = Per high power cell count ≤ 12
   = Moderate Degree= Per high powercell count 12 -20
   = Severe Degree = Per high powercell count > 20

2. Lymphocyte = Mild Degree = Per high power cell count 6-8
   = Moderate Degree= Per high powercell count 9 -11
   = Severe Degree = Per high powercell count > 11

3. Mast Cell = On special staining
   Mild Degree = Per high power cell count 0-1
   = Moderate Degree= Per high powercell count 2 -3
   = Severe Degree = Per high powercell count > 3

Table – XXIII
SHOWING POST THERAPY HISTOPATHOLOGY OF 30 PATIENT AFTER OLDER GROUP OF ANTI ALLERGIC DRUGS

S.No.	Structure	No. of Patient	Result			
			Complete response	Fair response	Poor response	No response
1.	Transitionala Epithelium	28	-	-	-	-
2.	Eosinophil Cell infiltration	18	-	3	7	8
3.	Edema	23	10	6	4	3
4.	Lymphocyte Cell infiltration	24	-	-	6	18
5.	Plasma Cell infiltration	6	-	-	2	4
6.	Mast Cell infiltration	7	-	-	2	5
7.	Squamous Metaplasia	2	-	-	-	2
8.	Fibrosis	5	-	-	-	5

Result

1. **Older group of antiallergic drugs**

   With older group of drugs, the edema and congestion improved 89.56% .

2. The eosinoehil count were reduce in 55.56% patient but majority of the patient having mild degree reduction there were mild change or no change in lymphocyte, plasma cell and mast cell count.

3. There were no change in fibrosis and squamous lining metaplasia.

Table – XXIV

SHOWING POST THERAPY HISTOPATHOLOGY OF 31 PATIENT AFTER NEWER GROUP OF ANTIALLERGIC DRUGS

S.No.	Structure	Pre T t No. of Patient	Post T t Result			
			Complete response	Fair response	Poor response	No response
1.	Transitional Cell Epithelium	30	-	-	-	-
2.	Eosinophil Cell infiltration	26	11	8	5	2
3.	Edema	25	16	7	2	-
4.	Lymphocyte Cell infiltration	21	10	7	4	-
5.	Plasma Cell Infiltration	12	7	3	2	-
6.	Mast Cell infiltration	9	2	4	3	-
7.	Squamous Metaplasia	1	-	1	-	-
8.	Fibrosis	3	-	-	-	3

Result

Newer group of antiallergic drugs

With newer group of drugs, the following result were obtained.

1. There were complite reduction of edema and congestion.
2. Lymphocyte count were completly reduced
3. Mast cell count reduced in Majority of patient but complete reduction were not present.
4. There were mild change in Fibrosis.
5. The Eosinophil count were reduced in 92.31%.

COMPLICATIONS

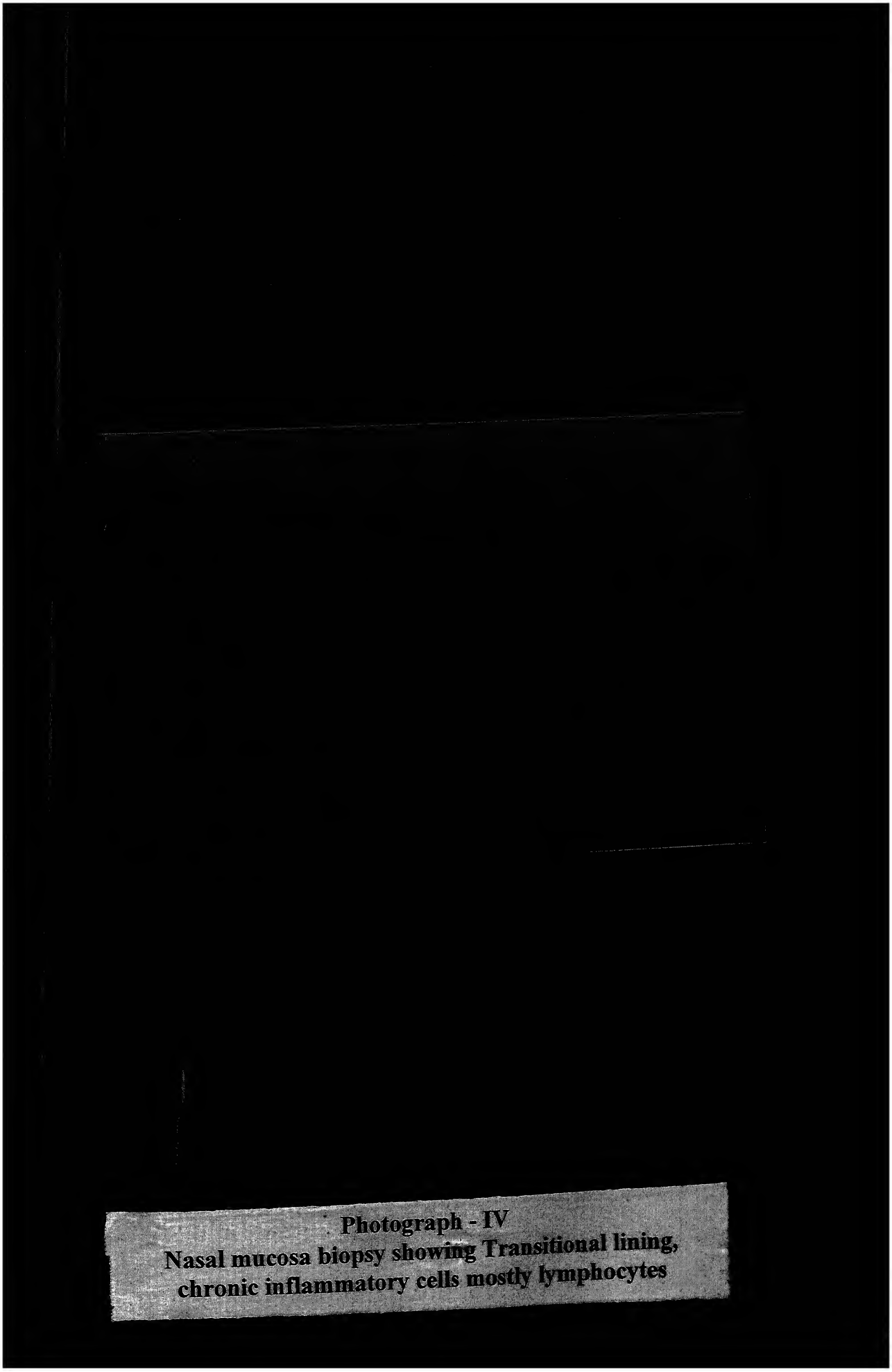
None of the patient in the present study had any complications except in 6 cases (9.30%) who had mild bleeding after the nasal biopsy that was controlled by anterior nasal packing and systemic heamostatics and antibiotic drugs.

AFTER EFFECTS

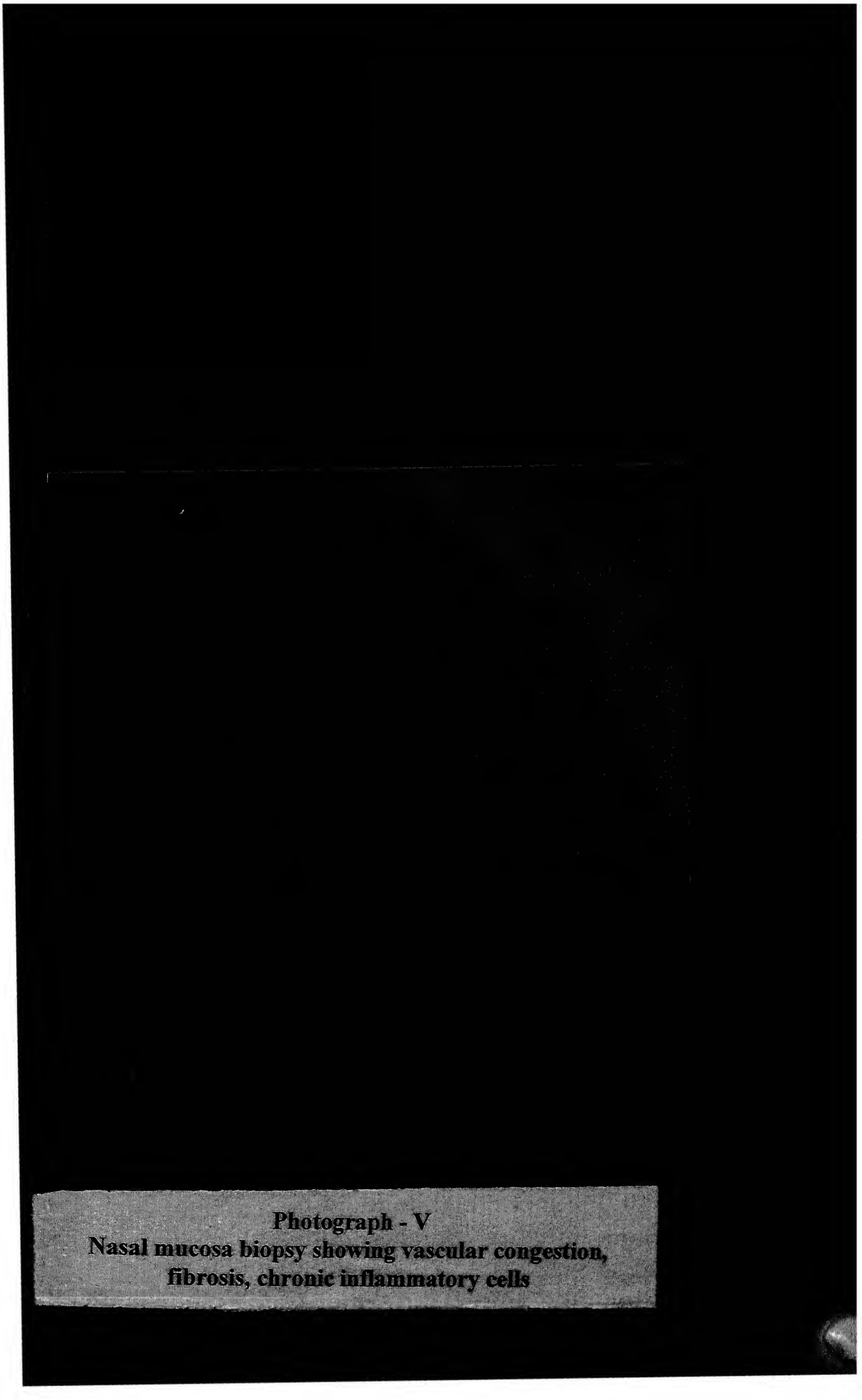
Few after effects were noticed in patients who had undergone older group of drugs like sedation, dryness of mouth, headach and rebound phenomenon etc., The newer group of antiallergic drug, no after effects were noticed except in one person mild epistaxis.

With topical xylomtazoiine therapy, 4 patients (23.53%) reported to have resistant rhinitis which was probably related to overuse of the drug, the condition known as rhinitis medicamentosa. Stinging discomfort or dryness locally in the nose were encountered infrequently.

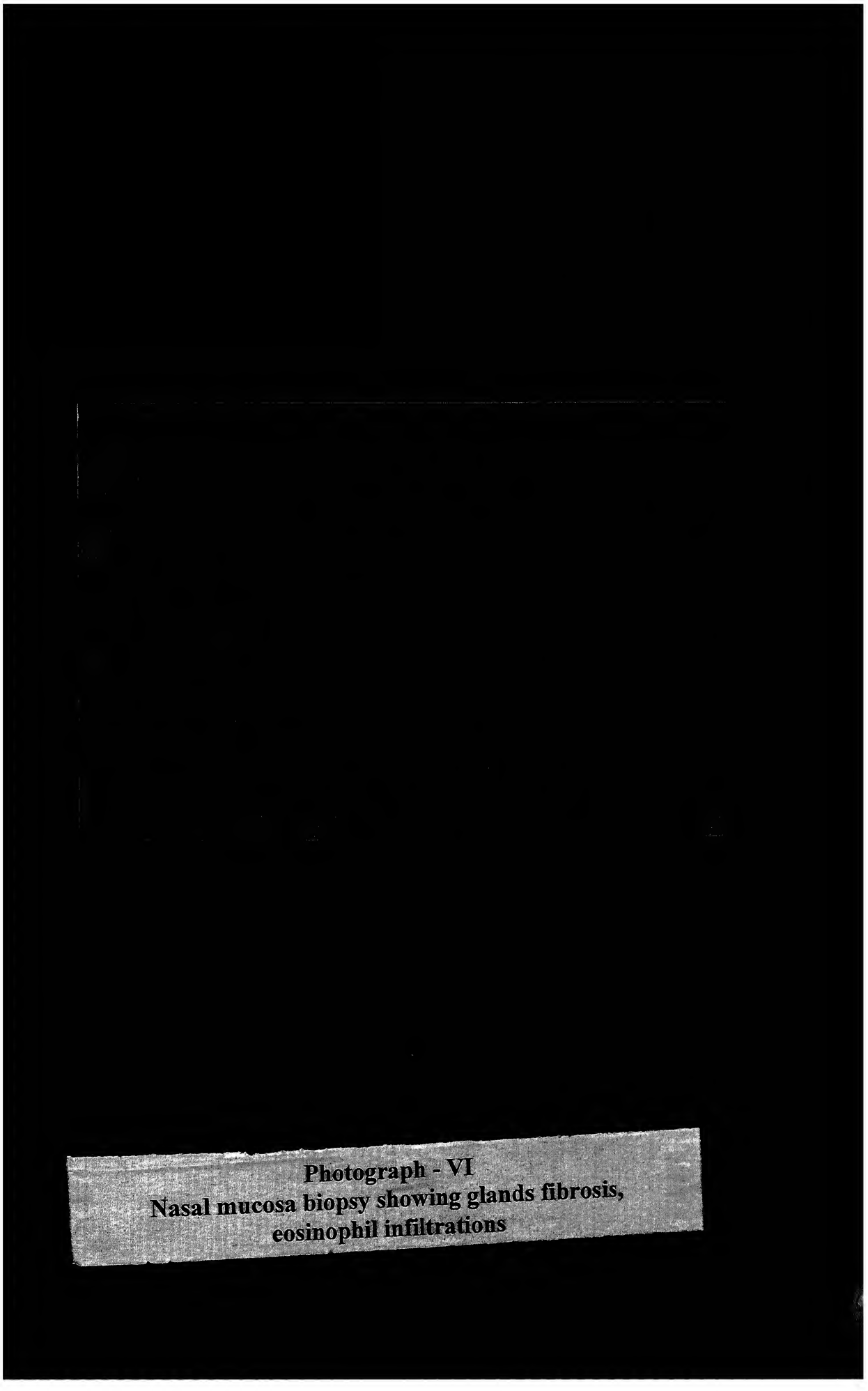
With topical budesonide therapy only one patient reported crusting and dryness of nose, on the other hand almost all the patients treated with budesonide felt such benefit that they continued the given treatment.



Photograph - IV
Nasal mucosa biopsy showing Transitional lining, chronic inflammatory cells mostly lymphocytes



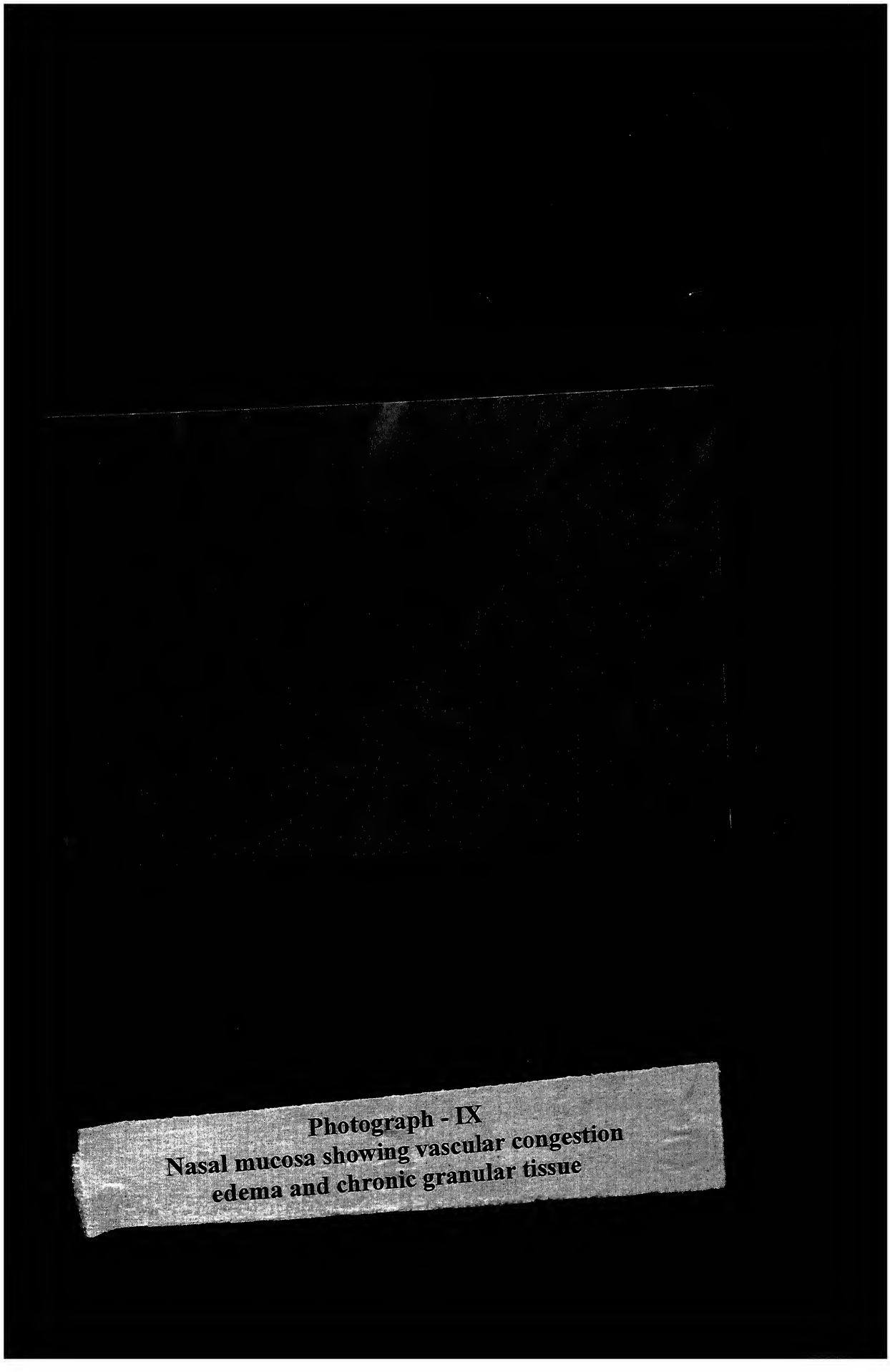
Photograph - V
Nasal mucosa biopsy showing vascular congestion, fibrosis, chronic inflammatory cells



Photograph - VI
Nasal mucosa biopsy showing glands fibrosis, eosinophil infiltrations

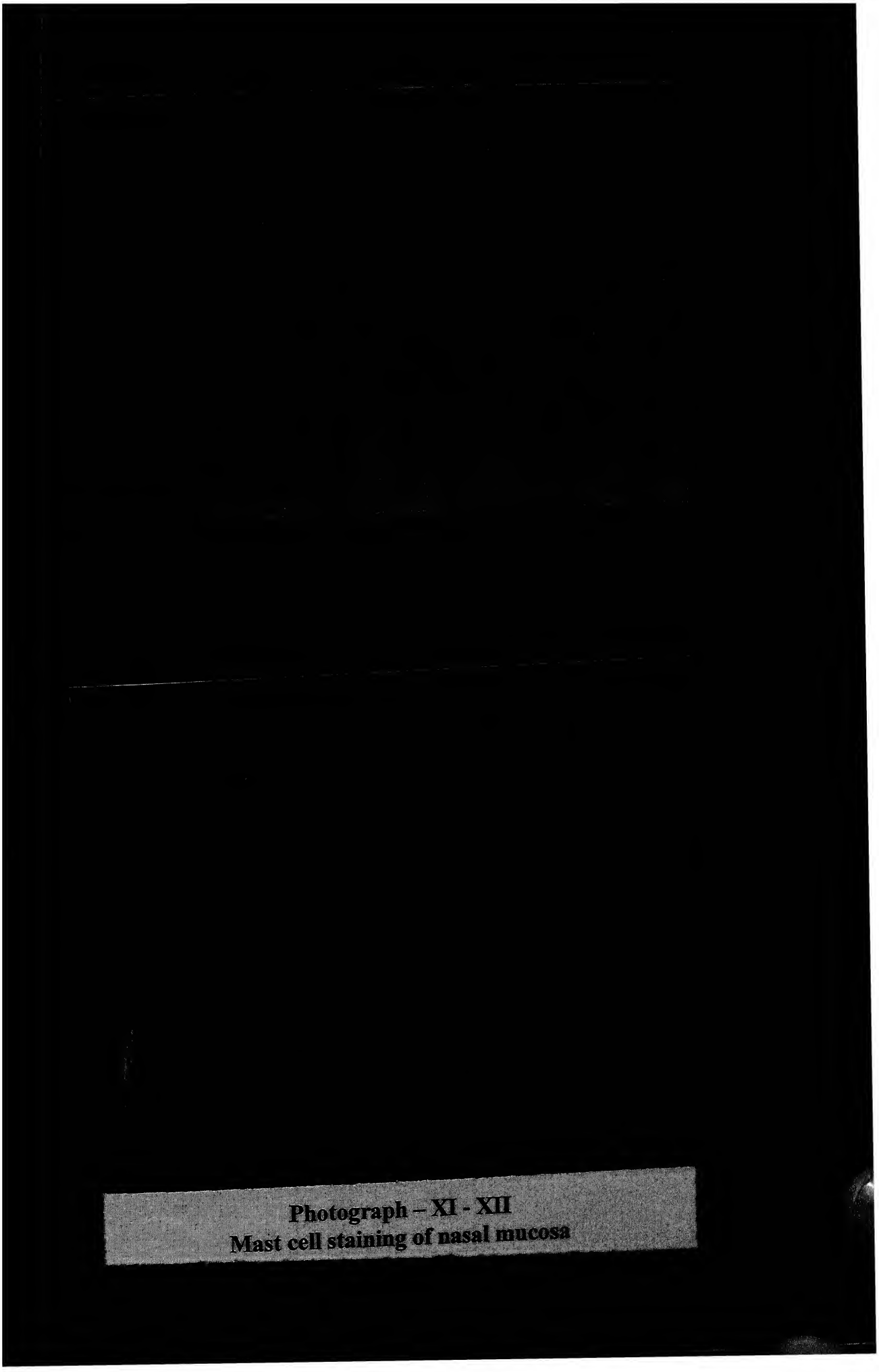
Photograph - VII
Nasal mucosa showing fibrosis with vascular congestion

Photograph - VIII
Nasal mucosa showing squamous metaplasia, granular tissue, blood vessels



Photograph - IX
Nasal mucosa showing vascular congestion edema and chronic granular tissue

Photograph - X
Nasal mucosa showing atrophic glands and congestion



Photograph – XI - XII
Mast cell staining of nasal mucosa

Discussion

DISCUSSION

The study comprises of evaluation of the comparison of the older and newer group of Antiallergic including oral and topical in 68 cases of allergic rhinitis attending E.N.T. OPD from July 2003 to September 2004.

AGE

Table - I

In this study most of the patients suffering from allergic rhinitis were in the age group of 10-30 years i.e. 45 cases (66.18%). This observation is in accordance with that of Negus (1955) hagy (1969) Lindquist et al (1986). The youngest patient was 13 years old and oldest 53 years of age. Most recent epidemiological studies clearly support this fact with data of a twofold or sometimes even threefold increase within the last decade (19-21). Prevalance figures between 1.3% and 52% in children have been recorded in different studies, meanwhile, other reports of older age groups described figures of 26% and 29% prevalence. This wide variation draws attention to the fact that allergy is the conjunction of hereditary predisposition and risk factors encountered in the environment. (Passali, Mosges et al., Consensus Conference of Allergic rhinitis in child hood).

SEX

Table - II

In the present study, allergic rhinitis was found to occur more in males as compared to females. Negus (1955) observed, both sexes were equally affected. The low incidence in female may be due to the fact that in our country; females are less exposed to outside atmospheric inhalants, allergens and temperature variations as they usually stay at home. but now this trend is gradually changed.

TYPE OF ALLERGY

Table - III

In our study in Bundelkhand region the perennial allergy is most common. The region could not be detected whether it is due to fungal, mite or any other cause because study was based on history. The seasonal allergic rhinitis was found mainly in winter season.

In Bundelkhand region the main allergen is parthenium pollen. The peculiarity of this pollen is that it occurs in large clump. Suba Rao et al., have work intensively on a Parthenium pollen and showed that reaginic allergy to this highly prevalent pollen was common in Banglore and Karnataka in general. Shivpuri and his colleagues in Delhi and Kabliwal in Jaipur were the pioneers in the work on allergy Shivpuri's group established that Helianthus, Amaranthus, cassia, Cenchrus, Morus, Imperata and other pollen were allergen.

OCCUPATION

Table - IV

In the present study incident of allergic rhinitis in relation to occupation revealed that house wives, office workers and students were the commonest sufferers. It is probable that these people are subjected to emotional stress, family troubles, examination worries and work load.

Wilson, 1955 said that allergic manifestations were most often seen in intellectuals or at least sofisticated people. The number of factory workers and labourers who are more exposed to inhalant allergens like dust etc. was less in this series. This is possible due to the fact they are unable to keep away from their work and attend the hospital regularly, so were not selected for study.

CAUSATIVE FACTORS

Table V

Physical allergy cases constituted the major group (51.02%) in the series. Most of patients were found to be sensitive to change in atmospheric temperature and cold. This high incidence of physical allergy demonstrates the importance of physical agents in the production of hypersensitivity symptoms. This factors has been stressed by Rose, (1940) and Henry Williams, (1944). Very recent data suggest that pre-exposure to a combination of nitrogen dioxide and ozone may amplify subsequent pollen-induced immediate symptoms of rhinitis (Rusnak et al., 1994).

In our study, in food allergy the most common allergy found with citrus fruits, milk, egg, curd & cashew nut. The pollen cross-reacting food contain proteins sharing epitopes with the pollen, these proteins are glycoproteins being heat and acid stable and thus not degraded in the stomach. Some patients were allergy to drug like aspirin and sulphonamide.

Every cell reacts with change in environment but the person who are allergic, they have more tendency to react with environment change like change in weather.

The smoke & Fumes probably act by following ways :

- It directly irritate the nasal mucosa.
- It causes cilliary damage & diminish cilliary movement.
- It disturb the sol and gel layer of mucous.

FAMILY HISTORY

Table VI

In the present study a positive family was obtained in 41.17% cases. James (1952) noticed such a hereditary factors in 50% of allergic rhinitis patients. Where as Gill, (1966) observed a positive family history was present in 46% case of allergic rhinitis the low incidence in this series may be because of ignorance or forgetfulness of patients or case selection. Genetic analysis of DNA from family members implicated genetic linkage with a gene (or genes) on chromosome 11q (Cookson et al. 1989). An exciting recent development is the colocalization on chromosome 11q of the gene for the high affinity IgE receptor, disorders of which, at least in part, may contribute to the atopic trait (Sandford et al. 1993).

PSYCHOLOGICAL FACTOR

The importance of psychological factors in the production of nasal and post nasal catarrh has been stressed by Fowler, (1950) in the present study moderate number of cases were anxious and depressed. It may be due to the disturbance in the balance of sympathetic, parasympathetic and Autakoid discharge.

SYMPTOMATOLOGY

Table IX – X - XI

In the present study classical symptoms of allergic rhinitis were, sneezing, (94.17%) discharge (64.70%) and nasal obstruction (75%) found amongst 68 cases in varying degree of severity. Among the other symptoms, recurrent attack of dull frontal headachem itching nose and eyes were the most important. Rinkel (1962) qualified recurrent headache as the most important neurological sign of allergy and itching as pathognomonic

sign. Lindquist et al (1986) studied 63 patients of allergic rhinitis and observed sneezing was the predominant feature (92.31%) followed by nasal obstruction 981.1%) and Rhinorrhoea (78%). The present study is in accordance with his observation.

SIGNS

Table IX- X - XI - XII

Shambough, (1945) stated that the typical allergic mucosa is pale and oedematous with increased secretion. He also found that in a typical case mucosa may be red and congested with tendency to dryness. The classical changes as stated by shambaugh were revealed in this series also. In the present study the nasal mucosa was pale bluish in (76.47%) cases. This observation is also in accordance with Bunnang et al, 1992.

The nasal discahrge was mostly watery in most of the cases. 42 cases (61.76%) of allergic rhinitis had watery discharge. This observation is in accordance with that of Binder (1984).

ASSOCIATED INFECTION

Incidence of infection of nose and sinuses was found to be nil in this series. While Hanset (1942) who has also worked on this aspect of allergic process, stimulated that definite sinus infection is comparatively rare in nasal allergy. Where as shambough, (1945) observed that at least 70% of chronic sin infection and 90% nasal infection have an underlying allergic factor responsible for chronicity.

Raised eosinophils (>10%) in blood were found in 34 (69.38%) cases. Neil Weir stated that peripheral blood eosinophil count can give information about the size of the "shock organ". If nose is the only organ affected the eosinophil count will usually be within normally limits.

HISTOPATHOLOGY

TABLE XVI-XXII –XXIII- XXIV

Each patient under gone either with older group of Anti allergic or newer group of anti allergic was subjected to nasal biopsy before the treatment. Histopathological picture in allergic rhinitis patients showed transitional multilayered hyperplastic epithelium, oedematous stroma, engorged blood vessels, eosinophil and mononuclear cell infiltration in most of the cases, depending on the stage at which patient reported for examination and treatment. These all findings are similar as described by successive workers, Hiranandani (1966), Weir (1967), Charles et al (1977), Bhargawa (1980), Lindquist (1986).

Post therapy biopsies of nasal mucosa revealed 73.33% (63.33% to 83.33%) improvement with older group of antihistaminic and 93.55% (90.32% to 96.79%) improvement with newer group of antihistaminic. Similar results were noted with budesonide by Pipkorm and Berge (1981), Lindquist et al (1986), Pipkora (1988). After topical treatment with Budesonide, significant decrease in no. of eosinophil has been observed. This is same as was noted by Klemi et al (1980).

In this series of 68 cases, only 61 patients received topical treatment and followed up regularly therefore only 61 patients were considered for assessment. Clinical and histopathological trial was conducted with two group of drugs used topically over nasal mucosa in 61 patients of allergic rhinitis. These drugs were older and newer. In this present study 30 patients were treated with older and 31 patients with newer drugs.

About (75%) of the patients of allergic rhinitis treated of older topical nasal drops had either total relief from their symptoms or had fair amount of relief from their symptoms.

About (93:56%) patient of allergic rhinitis treated with newer topically, had either total relief from their symptoms or felt a fair amount of relief from their symptoms.

TOPIAL TREATMETN WITH DECONGESTANT

With older topical drugs in allergic rhinitis nearly 2/3rd (73.33%) of patients under trial got significant amount of symptomatic relief very soon but 4 patients complained of rebound swelling. This finding suggests that nasal decongested relieves symptoms of allergic rhinitis rapidly and effectively but prolong use of it in the nose may lead to condition called rhinitis medicamentosa. Similar finding with xylometazoline has been reported by Petruson (1981), Fleece (1984), Akerlamd et al (1991), Graf et al (1994).

Despite of having slight over use and dose related side effect of rebound swelling or nasal stuffiness with xylometazoline, large number of patients (76.47%) got symptomatic relief with xylometazoline nasal drops or efcorlin. These clinical findings are in according in that of P.Graf and J.Juto (1994).

TOPICAL TREATMENT WITH NEWER ANTI - ALLERGIC

In ths present study of 31 cases of allergic rhinits, were treatd by newer topical drugs like budesonide, fluticasone and beclomethasone about (94%) patients of allergic rhinitis had either total relief from their symptoms or had a fair amount of relief from their symptoms.

Lindquist et al (1986) treated 63 patients of allergic rhinitis with budesonide. Our results as compared to his in the management of allergic rhinitis are nearly same.

Symptoms relieved	Good response	Fair response	Poor response
Sneezing			
Lindquist et al	72%	28.0%	-
Present study	76.92%	19.23%	3.84%
Nasal obstruction			
Lindquist et al	61.0%	31.0%	8.0%
Present study	85.71%	14.28%	-
Rhinorrhoea			
Lindquist et al	49.0%	43.0%	8.0%
Present study	70%	25%	5%

Bunnang et al. (1992) also reported their experience with Budesonide topically in the management of 33 cases of allergic rhinitis. The results of this study as compared to their study are follows :

Symptoms relieved	Good response	Fair response	Poor response
Nasal obstruction			
Lindquist et al	68%	28%	4%
Present study	85.71%	14.29%	-
Rhinorrhoea			
Lindquist et al	70%	25%	5%
Present study	66.67%	27.78%	5.55%

Wight et al (1992) managed 59 patients of allergic rhinitis with budesonide 400 micrograms and 800 micrograms topically per day. He observed more or less same benefit in the patients with either dose of

budesonide and no increase in adverse effects occurred with higher dose therapy.

Mc. Arthur (1995) carried out a comparative study budesonide and beclomethasone sprays in 88 adults with allergic rhinitis. In this study the results with Budesonide were good improvement in 69% of cases, fair in 22% of cases and there was poor improvement in 9% of cases.

To observation of the results of present study with Budesonide is similar to that of the other workers. A slight difference in the results may be attributed to the variation in number of patients under trial.

AU Lindqvist N. Balle VH Karma P. Karja. Lindstrom D. Makien J. Pukander J. Ruoppi P. Suonppa J. Ostlund W. et al. 1986 Apr. The long term safety and efficacy of budesonide nasal aerosol in perennial rhinitis. A 12 month multi centre study. Has been performed 104 patients with perennial rhinitis. The analysis revealed no histopathological change of the nasal mucosa. All nasal symptoms parameters assessed by the patient were significantly reduced from baseline during the follow - up of period. ACTH revealed no influence on the hypothalamic pituitary adrenal axis.

Otolarynogology – Head & Neck Surgery. 118 (5) 648-54, 1998 May. The tissue change associated with mometasone furoate use (200 microg/day) during a 12 month treatment period in patient with perennial rhinitis. Morphological examination of nasal biopsy specimens showed a decrease in focal metaplasia, no change in epithelial thickness, and no sign of atrophy after treatment. Immunocytochemical analysis of nasal biopsy specimens obtained before and after treatment revealed a significant decreased in major basic protein positive eosinophils and tryptase-positive mast cells in the epithelium and lamina propria after treatment and attenuate the inflammatory process by reducing the extend of inflammatory cells.

TI Assesment by nasal biopsy of long –· term use of mometasone furoate aqueous.

No recurrence in the symptoms of allergic rhinitis treated with Budesonide was noticed during the follow up period.

In the present study of allergic rhinitis cases we got more successful results with newer group of antiallergic drug (oral drug – levocetrizine, fexofenadine, ebastine) budesonide, Fluticasone and Beclomethasone (95.24%) when compared to (72.47%) in cases of xylometazoline, Naphazoline & Hydrocortisone topical spray. More-ever, there were almost no any side effects like rebound swelling or recurrence reported with Budesonide therapy, however the possibility of overuse related rebound Swelling, should be kept in mind before prescribing decongestant xylometazoline topically in allergic rhinitis patients. A Clear - Cut clinical efficacy evaluation can not be made in the present study as no control group was available. However, almost all the initially enrolled patients felt such benefit that they continued the given treatment. Another interesting observation was that there was no spontaneous increase in dosage. On the contrary several of the patients decreased their dosage and could still be free from nasal symptoms.

Summary and Conclusion

SUMMARY AND CONCLUSION

Allergy is defined as IgE medicated hypersensitity disease of muscus membrane of nasal airway characterized by sneezing, Itching, watery nasal discharge & nasal obstruction. It may be associated with allergic conjunctivitis & Bronchial Asthma. It may be seasonal, perennial allergic rhinitis. In Bundelkhand region most common allergic is grass pollen (Parthenium). In India the most common cause of allergic rhinitis is due to hous dust mite species D. Farinae. The characteristic feature of allergy is the preferential production of IgE antibody by human B Lymphocyte in response to antigen stimulation by common aeroallergens. The CD^{4+} Lymphocytes are essential for IgE production by B cell. In recent year two functionally different population of CD^{4+} helper cells have been recognized. The helper -1 (Th 1) subsent synthesizes & secretes IL-2 and IFN-γ where as Th^2 cells produce IL-4 & IL-5. The CD^{4+} Th^2 cells & Mast cell produce IL-4 & IL-5 are responsible for Ig E production. Allergen-IgE dependent activation of mast cell result in production of histamine, Tryptase, Bradikinin & Metabolic product of arachedonic acid by which PGD_2, LTB_4, C_4, D_4 (SRS-A) slow reacting substance are formed.

Immediate reaction of Allergy is produced by mainly Histamine via H_1 receptor. Hence H_1 antagonistic drug play important role in checking allergy.

From the present study, following conclusions were drawn :

(1) In this study the incidence of allergic rhinitis were found to be more below 30 years (66.18%). As the age advances, incidence sharply declines.

(2) In this study incidence of allergic rhinitis were found to be more in males (64.70%) than in females.

(3) Office workers and house wives were more affected than others (55.88%).

(4) In the present study the onset of disease was gradual in most of the cases.

(5) In the present study, it has been noticed that Physical allergy constitutes most important causative factor for the development of allergic rhinitis.

(6) In this study, family history of allergic disease was positive in only 41.17% cases.

(7) In tills study the chief symptoms with which the patients presented were sneezing (94.17%), Nasal Obstruction (75%) and less common symptom was rhinorrhoea (64.70%).

(8) In the present study the most common associated symptom was recurrent attack of common cold (66.17%).

(9) In this study the most common clinical findings observed on anterior rhinoscopy were pale bluish coloured nasal mucosa (76.47%) and mild to moderate enlargement of inferior turbinate (76.47%).

(10) In the present study majority of the patients (50%) suffering from allergic rhinitis had raised eosinophil count >10%.

(11) In this study the most common pre-therapy histopathological findings of the mucosa of inferior turbinate were transitional cell epithelium

(95.59%), oedematous stroma (70.59%) and eosinophil infiltration (64.7%).

(12) In the present study the effect of older group of antiallergic groups has been studied on the basis of relief of symptoms and change in histopathology and found to have complete response in 30% and fair response in 33.33% patients of allergic rhinitis. However, prolong use of it led to condition know as rhinitis medica mentosa or rebound swelling, in few patients.

(13) In the present study the effect of topical newer drugs has been studied in allergic rhinitis patients and found to have good response in 70.97% and fair response in 19.35% patients.

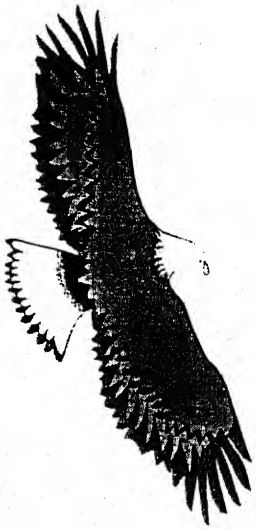
(14) In this study, poor response to therapy was more with older group of antiallergic then with newer group of allergic drugs.

(15) Comparing the post therapy clinical and histopathological results in this study, topical newer group of antiallergic drugs are found to be more effective and safe in the treatment of allergic rhinitis than topical older group of antiallergic drugs.

(16) The cost of newer group of drugs are very high then older group of drug while results differences is about 22% to 25%.

(17) The after effect of older group of drugs are mainly sedation with oral drugs and medicamentosa with topical drug on prolong used.

(18) We can used older group of oral antiallergic drugs as a first line in the poor patient, house wife's and the person who are not related to job which require concentration. Older topical group of drugs can be used for short period of time.



Master Chart

DETAILS OF CASES

PRE-THERAPY																						POST-THERAPY								
						SYMPTOMS				SIGNS			INVES.	HISTOPATHOLOGY							SYMPTOMS			HISTOPATHOLOGY						
S.No	Name	Age	Sex	Occupation	Family History	Sneezing	Nasal Obstruction	Rhinorrhoea	Other Ass. Symptoms	Turbinate Size	Colour of Mucosa	Discharge	D.L.C	Trans. Epi	Oedematous Stroma	Lymphocyte Inf	Eosinophil Inphil.	Mast Ceil Inf	Plasma Ceil Inf	Drug Used	Sneezing	Nasal Obstruction	Rhinorrhoea	Trans. Epi	Oedematous, Stroma	Lymphocyte Inf.	Eosinophil, Inf	Mast Ceil, Inf	Plasma Ceil, Inf
1	M.A	13	M	ST	+	+	+	+	+	GE	PB	WP	E12	TE	+	-	+	+	-	X. Ch	NR	CR	CR	TE	CR	-	FR	NR	NR
2	I.D.	15	F	ST	+	+	+	+	+	Mod E	PB	WP	E11	TE	+	+	+	-	+	X. Ch	FR	CR	CR	TE	CR	PR	FR	-	-
3	H.Z.	22	M	ST	+	+	+	+	-	Mil E	PR	WS	E13	TE	+	+	-	-	-	X. Ch	FR	CR	CR	TE	-	NR	NR	-	-
4	R.S.	45	M	LB	+	+	-	+	-	GE	PR	WS	E8	TE	-	+	+	+	-	X. Ch	FR	-	FR	TE	CR	PR	-	-	-
5	BA	33	M	OW	-	+	+	-	-	Mil E	PB	-	E5	TE	-	+	-	-	-	Edm	CR	NR	-	TE	CR	PR	-	NR	NR
6	KA	16	F	ST	-	+	+	+	-	GE	PB	WS	E11	TE	+	+	-	-	-	Edm	NR	FR	CR	TE	NR	NR	NR	-	-
7	NK	34	M	OW	+	+	+	+	+	GE	PB	WS	E12	TE	+	+	+	-	+	Edm	CR	NR	CR	TE	NR	NR	-	-	-
8	SJ	18	M	ST	+	+	-	-	+	Mil E	PR	-	E5	TE	+	+	+	-	-	Edm	CR	-	-	TE	FR	NR	-	NR	PR
9	DP	48	M	OW	-	-	+	+	+	Mod E	PR	WP	E14	TE	-	+	-	-	-	X. Ch	NR	FR	FR	TE	-	NR	NR	-	-
10	KF	32	F	HW	-	+	+	-	+	GE	PB	-	E8	SM	+	+	-	-	-	X Phe	-	CR	-	SM	PR	NR	PR	NR	NR
11	AK	20	M	ST	+	+	+	+	-	Mil E	PB	WP	E12	TE	+	+	+	+	+	X Phe	FR	NR	FR	TE	CR	PR	PR	-	-
12	RK	38	F	HW	-	+	-	+	+	No E	PB	WS	E11	TE	+	+	+	-	-	Xdm	NR	CR	NR	TE	FR	NR	NR	-	-
13	ZA	18	F	HW	+	+	-	-	-	Mil E	PR	-	E11	TE	-	-	-	-	-	E.Ch	CR	-	-	TE	CR	-	PR	NR	PR
14	SH	39	F	HW	-	+	+	+	+	Mil E	PB	WP	E14	TE	+	+	-	-	-	E.Ch	CR	FR	FR	TE	FR	NR	NR	-	-
15	MS	43	M	LB	+	+	+	+	+	Mod E	PB	WP	E15	TE	+	+	+	+	+	E.Phe	CR	CR	FR	TE	CR	PR	PR	NR	-
16	RT	32	F	HW	+	+	+	+	-	Mil E	PR	WS	E9	TE	+	+	+	-	-	X.Phe	FR	NR	NR	TE	-	+	NR	-	-
17	DM	34	M	FW	-	+	-	+	-	Mil E	PB	WP	E7	TE	-	+	-	-	-	X.Ch	NR	-	NR	TE	CR	PR	-	-	-
18	B	53	F	HW	+	+	+	+	+	GE	PB	-	E9	TE	+	+	+	+	+	E.Ch	FR	CR	CR	TE	NR	NR	-	NR	NR
19	LM	23	F	SW	+	+	+	-	-	Mil E	PB	-	E7	TE	+	+	+	-	-	E.Ch	FR	FR	-	TE	CR	NR	-	-	-
20	S	25	M	LB	-	+	+	+	-	Mod E	PB	-	E6	TE	+	+	+	-	+	E.Ch	FR	NR	FR	TE	-	NR	-	-	-
21	RK	27	M	LB	+	+	+	-	+	Mil E	PR	-	E11	SM	-	-	-	-	-	Edm	-	-	-	SM	FR	-	-	-	-
22	LN	28	F	HW	+	+	-	+	-	Mod E	PB	WS	E9	TE	+	+	+	+	+	Xdm	FR	CR	NR	TE	-	NR	FR	-	-
23	A	20	M	ST	-	+	+	-	+	Mod E	PB	WS	E16	TE	+	+	+	-	-	Xdm	NR	FR	FR	TE	FR	NR	NR	-	-
24	AKG	25	F	SW	+	+	+	+	-	Mil E	PR	WS	E12	TE	+	-	+	-	-	Xdm	FR	FR	NR	TE	PR	-	NR	-	-
25	JEA	23	F	OW	-	+	-	+	+	Mod E	PB	WP	E11	TE	+	+	-	-	-	Xdm	FR	-	FR	TE	PR	NR	PR	-	-
26	K	22	M	FW	+	+	+	+	-	Mil E	PR	-	E8	TE	+	-	+	+	+	X Phe	NR	FR	-	TE	CR	-	PR	-	-
27	A	51	M	LB	-	+	+	-	+	Mod E	PB	WS	E12	TE	+	+	+	-	-	E.Phe	CR	FR	CR	TE	PR	NR	PR	-	-
28	GP	21	F	OW	-	+	-	-	+	Mil E	PB	-	E8	TE	-	+	-	-	-	E.Phe	FR	-	NR	TE	FR	NR	PR	-	-
29	BD	23	M	LW	+	-	+	+	-	Mil E	PR	WP	E8	TE	+	+	+	-	-	E.Phe	CR	FR	FR	TE	-	NR	-	-	-
30	SA	17	F	OW	-	+	-	-	-	Mil E	PB	-	E6	TE	+	+	-	-	-	E.Phe	FR	-	-	TE	-	NR	NR	-	-
31	AJ	25	F	HW	+	+	-	+	+	Mod E	PR	WP	E14	TE	+	-	+	+	+	Xdm	NO FOLLOW UP								
32	WA	21	M	HW	-	+	+	+	-	Mil E	PB	WP	E12	SM	+	-	+	-	-	Xdm									
33	P	22	M	OW	-	+	+	+	-	No E	PR	-	E6	TE	-	+	-	-	-	E.Phe									
34	S	27	F	OW	-	+	+	+	+	Mil E	PR	M	E7	TE	+	+	-	-	-	X.Ch									
35	K	14	M	ST	+	+	+	-	-	GE	PN	WS	E8	TE	+	-	+	+	+	Flu.Lev	CR	CR	-	TE	CR	-	CR	CR	CR
36	N	15	M	ST	-	+	+	-	-	Mod E	PN	WS	E9	TE	+	+	+	+	+	Flu EB	CR	CR	-	TE	CR	CR	CR	CR	CR
37	KS	50	M	OW	+	+	+	-	+	Mil E	PN	WS	E9	TE	+	+	+	-	-	Flu Lev	FR	CR	-	TE	CR	CR	CR	-	CR
38	JA	45	F	HW	-	+	+	-	-	Mod E	PN	M	E10	TE	+	+	+	-	+	Bud EB	CR	CR	-	TE	FR	CR	CR	-	CR
39	PA	41	M	OW	+	+	+	+	+	GE	PB	WP	E16	SM	+	-	+	+	+	BECL FEX	CR	FR	FR	TE	CR	-	FR	FR	FR
40	VG	16	F	HW	-	+	+	+	+	GE	PB	WP	E18	TE	+	+	+	+	+	BECL EB	CR	-	CR	TE	CR	FR	CR	FR	FR
41	GR	17	M	ST	-	+	-	+	-	GE	PN	-	E9	TE	+	+	+	-	-	Flu Lev	FR	FR	FR	TE	CR	FR	FR	-	-
42	VA	18	F	HW	+	+	+	+	+	GE	PB	WP	E18	TE	+	+	+	+	+	Flu EB	CR	CR	-	TE	FR	CR	FR	FR	FR
43	PG	19	M	OW	-	+	+	-	-	Mil E	PB	WP	E12	TE	-	-	+	-	-	Bud Lev	NR	-	CR	TE	PR	-	CR	-	-
44	AS	19	M	LB	-	-	-	+	-	Mil E	PB	WP	E7	TE	+	+	+	-	-	Bud Lev	-	CR	CR	TE	-	FR	CR	-	-
45	AS	20	M	OW	+	+	+	+	-	Mil E	PN	-	E5	TE	-	+	+	-	-	Bud Fex	CR	CR	-	TE	-	CR	PR	-	-
46	AK	18	M	LB	-	+	+	-	+	Mil E	PB	WP	E6	TE	-	+	+	-	-	Bud Fex	CR	CR	-	TE	CR	CR	PR	-	-
47	H.Z.	22	F	HW	-	+	+	-	-	Mod E	PB	WS	E9	TE	-	-	-	-	-	BECL EB	FR	-	CR	TE	CR	CR	-	-	-
48	PL	25	M	OW	+	+	-	+	+	Mil E	PB	WP	E12	TE	+	+	+	+	+	BECL FEX	CR	CR	-	TE	FR	FR	FR	FR	PR
49	G	27	M	BM	-	+	+	-	-	Mod E	PN	-	E9	TE	-	-	-	-	-	Flu EB	CR	CR	-	TE	-	-	-	-	-
50	RA	28	M	ST	-	+	+	-	-	Mil E	PN	WS	E10	TE	+	+	+	-	+	Flu EB	CR	CR	-	TE	FR	FR	FR	-	PR
51	KK	22	F	HW	+	+	+	-	+	Mil E	PB	WP	E4	TE	+	+	+	-	-	BECL EB	FR	-	CR	TE	PR	FR	CR	-	-
52	AW	26	M	LB	-	+	-	+	+	Mil E	PB	WS	E16	TE	-	-	+	-	+	Bud Lev	CR	CR	CR	TE	PR	-	FR	-	CR
53	PA	27	M	OW	-	+	+	+	+	GE	PB	WP	E18	TE	+	+	+	-	-	BECL FEX	CR	CR	-	TE	CR	CR	CR	-	-
54	QT	30	M	BM	+	+	+	-	-	Mil E	PN	-	E6	TE	+	+	+	-	-	Flu Lev	CR	CR	CR	TE	FR	CR	PR	-	-
55	RS	24	M	OW	-	[illegible]		+	-	Mod E	PN	-	E5	TE	-	-	-	-	-	BECL EB	-	-	CR	TE	FR	-	-	-	-
56	AJ	23	M	ST	-	+	+	+	+	Mil E	PB	WP	E13	TE	-	+	+	-	-	BECL FEX	FR	FR	FR	TE	-	FR	FR	-	-
57	NS	21	F	HW	+	+	+	+	-	Mil E	PB	WS	E14	TE	+	+	+	-	-	Bud Fex	CR	FR	CR	TE	CR	CR	CR	-	-
58	SK	22	M	OW	-	+	-	+	-	Mil E	PN	-	E10	TE	+	+	+	-	-	BECL Lev	CR	-	-	TE	PR	FR	FR	-	-
59	MK	24	F	HW	+	+	+	-	-	No E	PN	-	E8	TE	+	-	+	-	-	Flu Fex	CR	CR	CR	TE	PR	-	NR	-	-
60	MA	25	M	ST	-	+	+	+	+	Mil E	PB	WP	E16	TE	+	+	+	+	+	BECL Lev	CR	CR	CR	TE	FR	PR	NR	PR	CR
61	DA	21	F	HW	-	+	+	+	+	Mil E	PB	WS	E18	TE	-	+	+	+	-	Flu Fex	CR	CR	CR	TE	FR	PR	CR	PR	-
62	KJ	40	M	FW	-	+	-	+	-	No E	PB	WP	E6	TE	+	+	-	-	-	Bud EB	NR	-	NR	TE	NR	FR	-	-	-
63	SS	31	F	HW	-	-	-	+	+	Mod E	PB	WS	E8	TE	+	+	+	-	-	Flu Lev	FR	-	CR	TE	NR	PR	PR	-	-
64	AS	32	M	FW	-	-	+	+	+	Mil E	PB	WS	E5	TE	+	+	-	-	-	Bud Lev	-	CR	CR	TE	CR	PR	-	-	-
65	MI	35	M	OW	-	+	+	+	+	Mil E	PB	-	E6	TE	+	-	+	+	+	BECL FEX	-	CR	FR	TE	-	-	PR	PR	CR
66	AI	33	M	BM	-	+	-	-	-	Mil E	PN	-	E12	TE	-	+	+	-	-	Flu FEX	NO FOLLOW UP								
67	KK	36	M	FW	-	+	-	-	-	Mod E	PB	-	E11	TE	+	+	+	-	+	BECL FEX									
68	NS	39	M	LB	-	-	+	+	-	Mod E	PB	WS	E12	TE	+	+	+	-	-	Flu Fex									

ABBREVIATION

M= Male; F=Female; ST= Student; HW=House Wife; OW=Office Worker,LB=Labourer; BM=Businessman;FW=Factory Worker; +=Positive; -=Negative; GE=Gross Enlargement

ModE=Moderate; MilE=Mild Enlagement; NoE=No Enlargement; PB=Pale Blue; PR =Pink, WP=Watery Profuse; WS= Watery Scanty; M=Mucoid; TE=Transitional Cell Epithelium; SM=Squamous Metaplasia E=Eosinophil

CR=Complete Response; FR=Fair Response; NR=No Response; PR=Poor Response

X=Xylocaine; E=Efcorlin; Flu=Fluticasone; Bud=Budesonide, BECL=Beclomethasone; Ch= Chlorpheniramine dm=Dimethendine; Phe=Pheniramine; Fex=Fexofenadine, Lev=Levocetrizine; EB=Ebastine

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